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of the

INDIAN MEDICAL ASSOCIATION

VOLUME 25 NUMBER 12 CALCUTTA NOVEMBER 16, 1955

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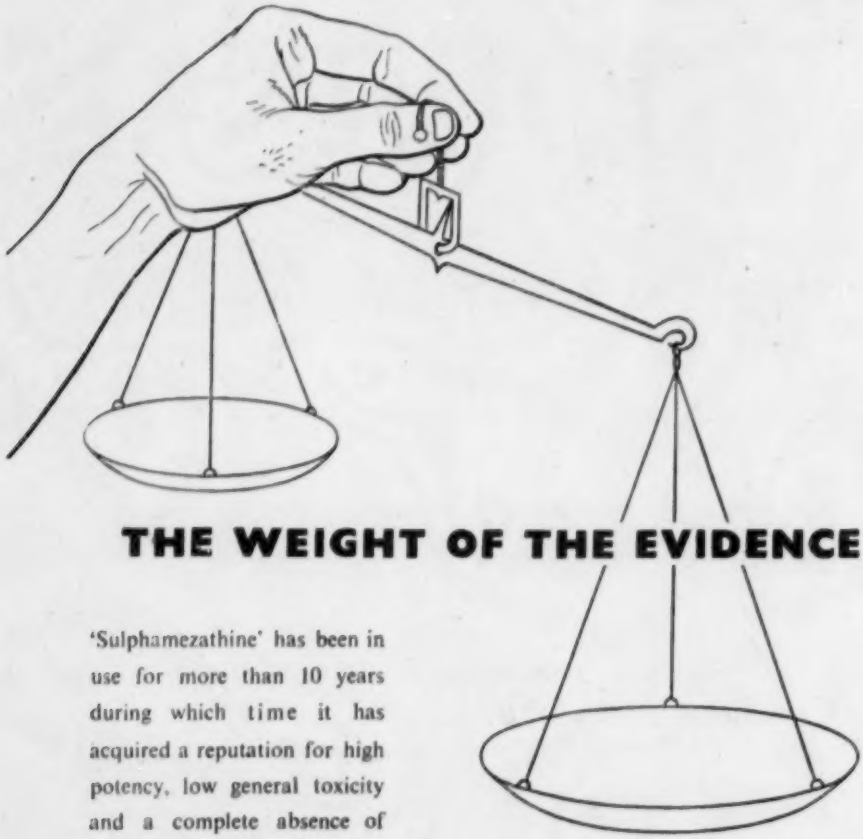

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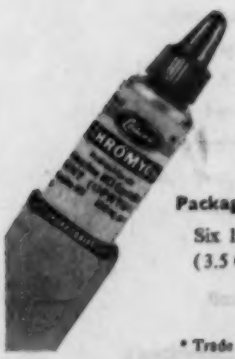

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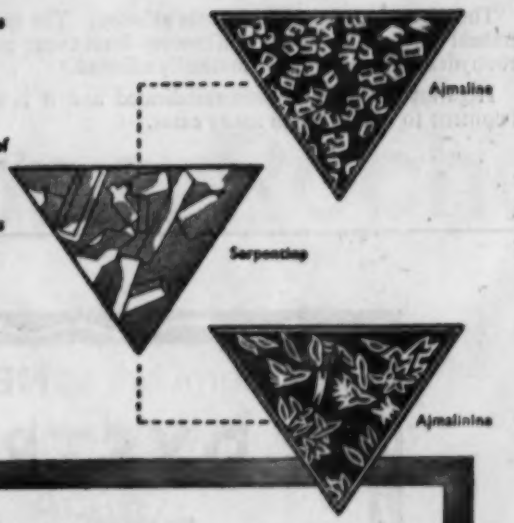

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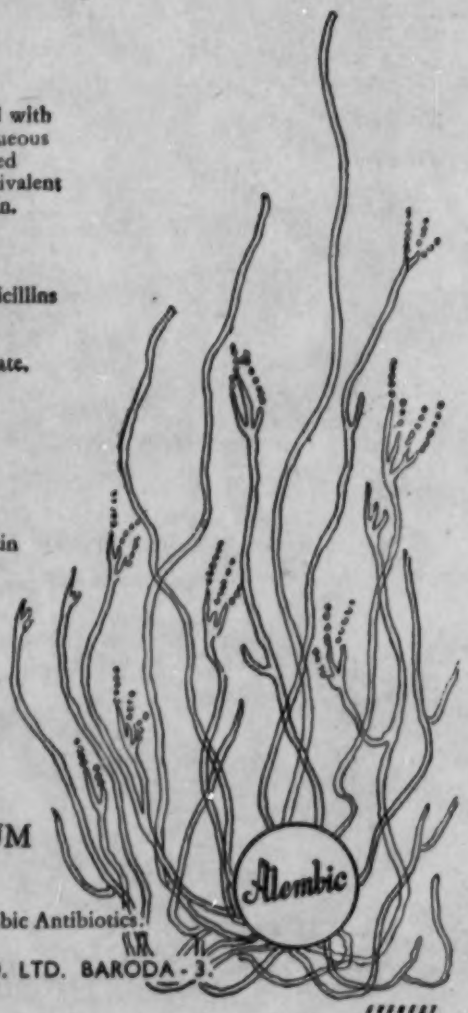

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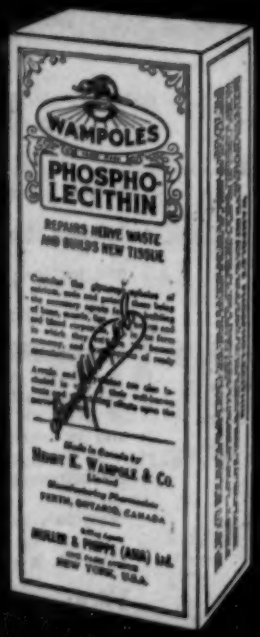

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Journal of the Indian Medical Association

VOL. 25, No. 12 — CALCUTTA — NOVEMBER 16, 1955

PUBLISHED TWICE A MONTH

ORIGINAL ARTICLES

HURTHEL-CELL TUMOURS OF THE THYROID

D. J. REDDY, M.D., M. MUNISWAMY, B.A., M.S.,
D. BHASKHARA REDDY, M.D. AND D. N. APPA RAO, M.B.B.S.,

Departments of Pathology, Guntur and Andhra Medical Colleges.

Adenomatous goitres of the thyroid are frequently encountered by the clinician. Hurthel-cell adenoma or its malignant variant is a less common neoplasm of the thyroid and a preoperative diagnosis of the same is rarely made. The pathologist not infrequently finds 'Hurthel cells' composing exclusively an adenoma or a carcinoma and sometimes as clusters of cells in normal thyroid itself. These tumours continue to excite interest to the medical profession from varied view points of aetiology, pathology and treatment.

The large cells with finely granular eosinophilic cytoplasm that compose these tumours were originally described by Baber (1881) although Ewing (1928) credited Hurthel as the author of its original description in designating them as 'Hurthel-cell tumours' for the simple reason that Hurthel (1894) demonstrated these cells in the thyroids of young dogs. The exact nature and significance of the cells forming these tumours have still not transgressed the stage of theorization, although some are of opinion that it is a cellular type of thyroid involution.

The exact incidence of this neoplasm is difficult to evaluate from the published reports since some have reported them under adenoma while others have included them under carcinoma. Many view these growths as malignant and in the manual of tumour nomenclature edited in 1951 they are designated as 'Hurthel-cell carcinoma'. Goldenberg (1953) observed that Hurthel-cell tumours formed 4·5 per cent of the solitary adenomata in 1330 thyroid biopsies while Chesky *et al* (1951) reported 5 per cent incidence. Incidence of malignancy in these tumours is variable according to the published reports for Goldenberg (1953) recorded 30 per cent while Frazzel and Duffy (1951) found 10 per cent incidence among the thyroid tumours reviewed by them. Horn (1954) observed that in 61 Hurthel-cell tumours 34 were malignant. The variation in the incidence of malignant Hurthel-cell tumours is probably attributable to the thoroughness exercised in screening the histological sections, the number of sections screened, the criteria employed for grading of malignancy and the follow-up of the operated cases. Analysis of the thyroid biopsies of the pathology departments of Madras and Andhra Medical Colleges is given in Table 1.

TABLE 1—SHOWING AN ANALYSIS OF THE THYROID BIOPSIES OF MADRAS AND ANDHRA MEDICAL COLLEGES

	Adenoma—simple		Hurthel-cell adenoma		Hurthel-cell carcinoma		Toxic goitre		Lymphadenoid goitre	
	No.	per cent	No.	per cent	No.	per cent	No.	per cent	No.	per cent
Andhra Medical College ...	97	84·3	—	—	1	0·87	11	8·6	6	5
Madras Medical College ...	127	86	6	4·9	—	—	12	8	2	1·3

There is nothing characteristic about the gross pathology of these neoplasms and are often indistinguishable from benign adenomata. They are invariably encapsulated. The distinct cytology of the growth makes the pathologist recognise them as a distinct entity. The cytological picture is not always typical of malignancy and the spotting of venular invasion points to malignancy and not cell variation. The cells composing the tumour may be arranged in trabecular, alveolar, papillary or large follicular patterns.

The rarity of published reports of Hurthel-cell tumours in our country and their clinical importance offered incentive to record the clinico-pathological findings of three cases studied by us.

Case Reports

Case 1—N.R., aged 25, was admitted on 2-7-1954 in the Government General Hospital, Guntur, for dysmenorrhoea for which dilatation and curetting were done. A circumscribed swelling was observed on the left half of the neck. No thyrotoxic symptoms were complained of by the patient. Adenoma of the thyroid was diagnosed. The tumour was enucleated on 20-7-1954 by a collar incision under local anaesthesia. Postoperative period was uneventful.

Morbid anatomy and histology [Biopsy No. 18/54]—Comma-shaped swelling. The circulo-ovoid tumour mass was well defined and sectioned surface was homogeneous, of whitish colour and capsulated (Fig. 1, *vide* Plate). Sections from multiple blocks revealed lobules made up of large cells with eosinophilic cytoplasm and discrete nucleus (Fig. 2, *vide* Plate). The pedicle of the tumour contained papillary adenoma (Fig. 3, *vide* Plate). Cytological evidence of malignancy was lacking in all the sections studied. The tumour tissue did not infiltrate the capsule or veins.

Morbid anatomical diagnosis—Hurthel-cell adenoma.

Case 2—M., female, aged 27, was admitted into Sanjivi Clinic for lemon-sized tumour in the midline of the neck (Fig. 4, *vide* Plate). She had it since 4 years with fluctuations in the size of the tumour.

No thyrotoxic symptoms were complained of by the patient.

Clinical diagnosis—Cystadenoma thyroid.

Under basal and local anaesthesia enucleation of the growth was done.

Morbid anatomy and histology [Biopsy No. 81/54]—Bits of the tumour tissue were only available for histological study. Sections revealed typical Hurthel-cells arranged in alveolar pattern and in some places in cord like pattern. Clusters of these cells were seen in tiers around blood vessels, presenting a peritheliomatous pattern. Cytological evidence of malignancy was wanting.

Morbid anatomical diagnosis—Hurthel-cell tumour of the thyroid.

Case 3—G., male, aged 30, was admiitted in King George Hospital, Visakhapatnam on 21-12-1953 for a swelling in the neck of 2 years' duration. This swelling did not cause any disability. The skin was dry. The skin over the swelling was not adherent. The swelling was freely movable. Trachea was pushed to one side to a slight extent. Regional lymph nodes in the neck were not palpable. Pulse—78 per minute, B.P.—130/90 mm. of Hg., B.M.R.—11 per cent, fractional test meal showed hyperacidity.

Clinical diagnosis—Adenoma thyroid.

On 19-1-1954 a large adenoma was removed from the left lobe of the thyroid.

Morbid anatomy and histology [Biopsy No. 7976]—Well circumscribed, smooth avascular tumour of 3 inches by 2 inches in size. Sectioned surface was of greyish white colour with spots of cystic degeneration. The mass was encapsulated (Fig. 5, *vide* Plate). Multiple sections studied from several blocks gave varying cytological picture. In places the cells appeared to possess no features characteristic of malignancy and they were arranged in cord like pattern. Typical Hurthel-cells were seen arranged in alveolar pattern, each group of cells was separated from the other by thin connective tissue. These cells varied in size and were hyperchromatic. Mitotic figures were not infrequent (Fig. 6, *vide* Plate). In places follicular arrangement of the cells were seen, the lumen in most places was devoid of colloid. The cells bordering the lumen were deeply stained (Fig. 7, *vide* plate). Massive zones of necrosis were observed. The cytological features seen in the sections bear indisputable evidence of malignancy.

Morbid anatomical diagnosis—Hurthel-cell carcinoma.

Comment

The variation in incidence of Hurthel-cell tumours observed in the Madras and Andhra Medical Colleges is in a large measure due to the personal factor of the pathologist screening the sections. All the three cases were pre-operatively recognised as adenoma and the true Hurthel-cell nature of the neoplasms was disclosed only on biopsy examination. The last case showed indisputable evidence of malignancy, though malignancy could never have been recognised clinically. This should warrant the surgeon to submit every operated material of thyroid for biopsy examination. Follow up of all cases reported by the pathologist "Hurthel-cell adenoma or carcinoma" is most essential and desirable since no one could predict with any degree of certainty when the adenoma could acquire aggressive qualities and the tumour either would recur or set distant metastases at a future date. Though there is a definite tendency for the surgeon and the pathologist to include all tumours of the Hurthel-cell type under carcinoma, we feel that we cannot but retain the terms adenoma and carcinoma as long as one observes the cytological features of either. We can only hint to the surgeon that Hurthel-cell adenomata are to be deemed potentially malignant.

Fig. 1 (Case 1)—Showing homogeneous sectioned surface of the tumour nodule with an intact capsule.

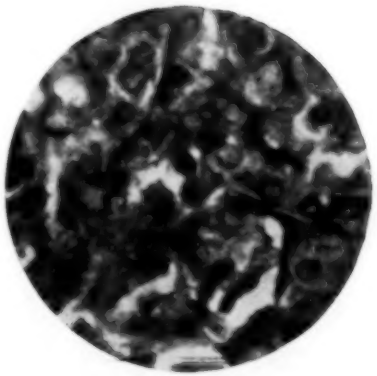

Fig. 2 (Case 1)—Photomicrograph showing large cells with granular cytoplasm and conspicuous nucleus. (H. & E. × 100)

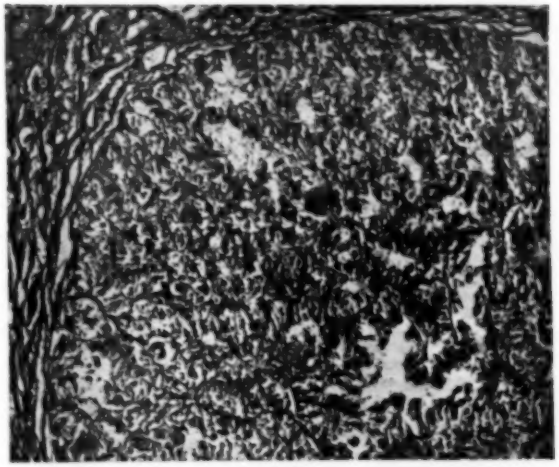

Fig. 3 (Case 1)—Photomicrograph showing papillary adenoma surrounded by fibrous tissue capsule. This structure was observed from the tissue of the tail end of the tumour mass. (H. & E. × 60)

Fig. 4 (Case 2)—Photograph showing circumscribed nodule in the median line of the neck.

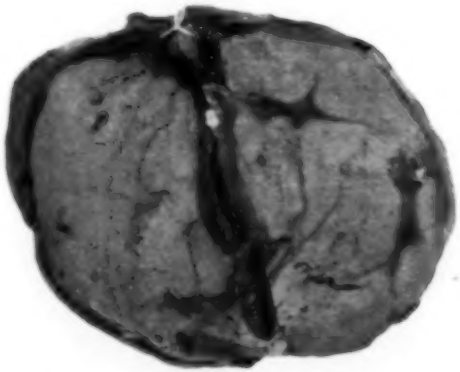

Fig. 5 (Case 3)—Sectioned surface of the neoplasm bordered by an incomplete capsule and showing cystic spaces.

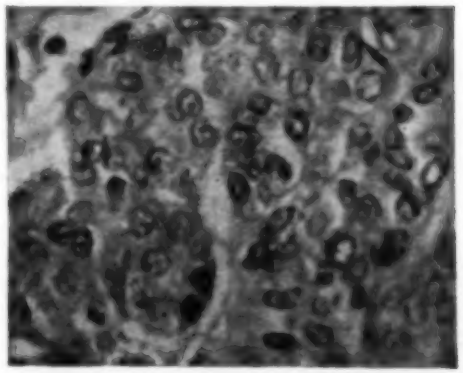

Fig. 6 (Case 3)—Photomicrograph illustrating alveolar arrangement of tumour cells which show variation in size and many are deeply stained. (H. & E. × 100)

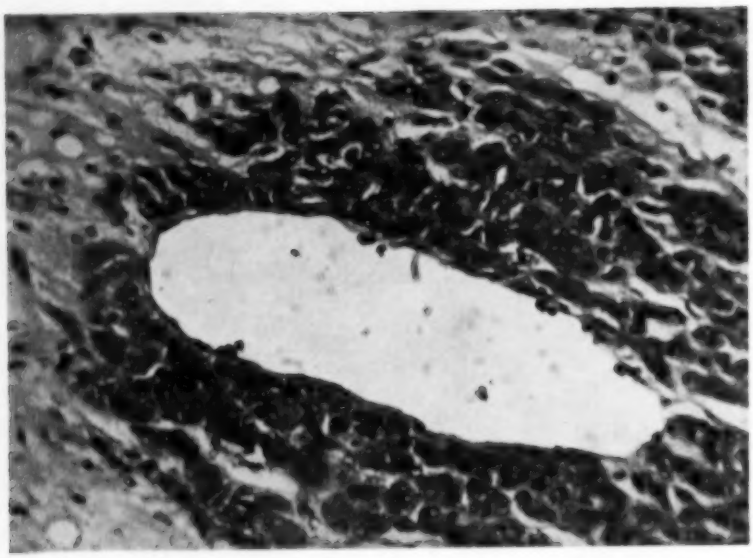

Fig. 7 (Case 3)—Photomicrograph illustrating follicular pattern of the cells. Many of the cells are hyperchromatic. (H. & E. × 60)

REDDY ET AL—*Hurthel-cell Tumours of the Thyroid (pp. 465-69)*

Fig. 1

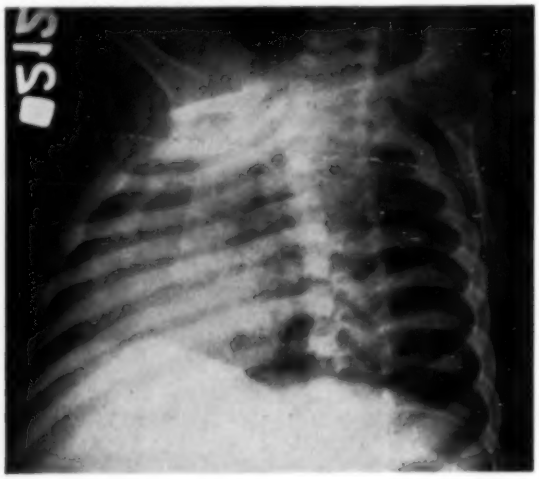

Fig. 2

GHOSH AND BISWAS—*Incidence of Congenital Cardiac Anomaly in Mongols with a Case Report of Fallot's Tetralogy in a Mongol (pp. 472-475)*

ENGRAVED & PRINTED BY REPRODUCTION SYNDICATE, CALCUTTA 6

SUMMARY

Literature regarding Hurthel-cell tumours of the thyroid is briefly reviewed.

Clinicopathological findings in two cases of adenoma and one case of carcinoma of Hurthel-cell type are recorded. Stress is laid on the necessity for postoperative follow up of these cases since a considerable percentage of the tumours reported adenoma at any time may acquire malignant features.

There is still a need to retain the terms adenoma and carcinoma to tumours of Hurthel-cell type.

ACKNOWLEDGMENT

We are grateful to Dr. D. Govinda Reddy, Professor of Pathology, Madras Medical College and Dr. G. S. Viswanathan, Andhra Medical College for placing the biopsy records at our disposal for analysis. We are grateful to Drs. C. K. P. Menon and K. Subba Rao for the clinical histories. We acknowledge with pleasure the help rendered by the photoartist Mr. Edwin.

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HELPFUL EXPERIENCES WITH INTESTINAL OBSTRUCTION

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Intestinal obstruction is possibly the commonest cause of acute abdomen seen in India. It is reported that in Massachusetts General Hospital the frequency, considering all other surgical cases, is only 0·85 per cent. However, it is much more frequent than that, in Miraj. In fact, it is seldom that we do not have at least one case in the wards. Probably it is as frequent an emergency in India, as acute appendicitis is in Western countries.

While taking the patients' history, the only precipitating factor that we have been frequently able to elicit is that bowel stoppage followed an extra-large meal—perhaps a wedding feast—where many *Jondhla bhakar* (jowar breads) had been consumed. With a history of bowel stoppage, a careful physical examination, including digital examination of the rectum is of course a prime requisite. One or more enemas should be given no matter what may be said regarding previous enemas. When the enemas are not productive, a silent, tender and tense abdomen, with or without visible peristalsis, clinches the diagnosis and the patient should be prepared, mentally and physically, for operation. On opening the abdomen, we frequently find adhesions, caused by tuberculous peritonitis or constrictions due to tuberculous enteritis. Adhesions also result apparently from enteric fever, pelvic disease and appendicitis. Occasionally they follow intestinal perforation by *Ascaris lumbricoides*.

For many years one of the authors (E.H.E.) has been deeply concerned with this problem, since quite a number of such cases have fallen to his lot to treat. The immediate impetus, however, came from a statement by Anderson (1954) of Ahmednagar, that the over-all mortality for intestinal obstruction cases treated by him over a period of five years, was 31 per cent. (2) Like a number of others who heard this statement, I thought, "Surely that is a higher percentage of mortality than ours!" At any rate, we decided to study our records. The present study is that of all of the cases of intestinal obstruction treated by all of the surgeons operating at the Miraj Medical Centre during the period January 1, 1940 to October 31, 1954. To illustrate this briefly and graphically, three tables of figures have been prepared.

Table 1 shows that strangulated hernias are by far the greatest single cause of obstruction: 113 out of a total of 431. Next in order comes volvulus, accounting for 16 per cent of our cases. Usually there are many cases of volvulus in any series of bowel obstruction cases, sometimes as high as 60 per cent (Anderson, 1950). Adhesions, bands, kinks and constrictions have been lumped into one class since not infrequently an obstruction is caused by one or a combination of these and it is impossible to determine with certainty which are the more important factors. *Imperforate anus* cases have been recorded since 1st January 1950. There were 14 male and 4 female babies, a total of 18 cases. Of these 12 were cured and 4 died or were discharged unimproved. For 2 cases no result was recorded. For the 16 cases then, where a result was recorded, there is a record of 12 recoveries or

TABLE 1—SHOWING VARIOUS CAUSES OF OBSTRUCTION

	Number of cases	Per cent
Hernias : Strangulated and incarcerated	113	26·20
Volvulus	71	16·45
Adhesions 59		
Bands and kinks ... 19	92	21·52
Constriction (including T.B.) ... 14		
Lumbricoids	31	7·19
Perforation	22	5·08
Intussusception	5	1·12
Diverticula	4	0·92
Other causes	12	2·74
Not determined	81	18·78
Total ...	431	100·00
Cases of imperforate anus (Since 1st Jan. 1950)	18	

TABLE 2—SHOWING SEX AND AGE GROUPS

Age in years	Number of cases	Per cent
1 to 10	52	12·1
11 „ 20	39	9·4
21 „ 30	101	23·4
31 „ 40	80	18·36
41 „ 50	91	21·20
51 „ 60	45	10·49
61 and above	23	5·26
Total ...	431	100·00
Males	341	79·11
Females	90	20·89
Total ...	431	100·00

TABLE 3—SHOWING THE NUMBER OF CASES AND THE RESULTS OF TREATMENT DURING THE THREE DIFFERENT FIVE-YEAR PERIODS

5 Year Period	No. of cases	Cured	Improved	Unimproved	Died
1940—1944 ...	176	78 (44·29%)	17 (9·15%)	10 (5·67%)	71 (40·39%)
1945—1949 ...	139	75 (53·96%)	15 (10·89%)	2 (1·43%)	47 (33·81%)
1950—1954 ... (End of October)	116	60 (51·72%)	16 (13·79%)	6 (5·17%)	34 (29·32%)

recovery in 75 per cent of this small series. Other causes, lumbricoides, perforation, intussusception, diverticula, etc., are noted in diminishing percentages. The large figure, eighty one (81), in the category of "cause not determined" is partly because of the number of cases whose condition was too poor for operation. It is also, partly it must be admitted, a silent commentary on the incompleteness of the records.

Table 2 shows frequency by age and sex groups. The greatest frequency is in the age groups between 20 to 50 years—A disease of early life. Males constitute 79 per cent and females 21 per cent. Just why there should be such a great predominance of males, it is difficult to say, although Anderson (1954) feels, the broader female pelvis may be an important factor.

Table 3 shows the number of cases in the three different five year periods and the result of the treatment of these cases. The number of cases, for some unknown reason, have markedly diminished in the last 15 years, being 176 in the first five year period, 139 for the second and 116 in the last—a diminution of approximately 33 per cent. For this reason, the apparent diminution of deaths from 71 to 34 or approximately 50 per cent is not authentic, as it at first appears to be. Actually our total number of in-patients has diminished 6·6 per cent in the same period. Let us look at the percentages :

Table 3 also shows the percentages for the same series of cases (176, 139 and 116). The percentages for "Cured" cases improved from 44·29 per cent in the first five year period to 51·72 per cent in the most recent period. It should be explained that when a patient was discharged "Improved" he was likely not fully recovered. He might return to the Out-Patient Department for some days for "tonic" treatment or for dressings to an incompletely healed wound. The category of 'Unimproved' cases includes those who were too weak for operation or those whose condition was for other reasons deteriorating and whose relatives chose to take them home before death. Therefore, to actually give a cleaner picture of results, we think it would be desirable to combine the percentages for "Cured" and "Improved" cases. This would give a total of 53·94 per cent for those which could properly be considered "Recovered" cases in the first five year period and 65·57 per cent for the last five year period.

It is also to be noted that the percentage of deaths decreased from 40·39 per cent in the first period to 29·32 per cent in the most recent one. This means that mortality rates decreased over a 15 year period by 27·40 per cent.

Our figure of 29·32 per cent mortality is slightly less than Anderson's 31 per cent and is for approximately the same years 1950-54. The difference, however, we feel to be insignificant. The

fact remains that intestinal obstruction is a very serious condition in spite of all the aids modern science has given us.

What are the causes of this increase in recovery and decrease in mortality rates? In a paper of this kind, the briefest of resumes is all that space will permit. First should be mentioned the restoration of the balance of fluids and electrolytes by the intravenous infusion of glucose and saline, which, of course is far superior to the older methods of proctoclysis and hypodermoclysis (Conn, 1950). Second, we would mention the passage of the duodenal tube or the Miller-Abbott tube. This and intravenous infusion is started immediately on admission. A tube of one of these types is connected with a gravity suction apparatus of the Wangensteen type (Wangensteen, 1949) and often results in a prompt and almost dramatic lessening of upper abdominal distension. In the rare case the lessening of abdominal tension actually effects a removal of the cause of the obstruction and the patient, after a short time, passes gas and faeces by rectum. While such a result is most gratifying, we have learned not to delay operation in the hope that this may occur, but to operate as soon as the patient has attained an optimum pre-operative condition, usually after he has received between 500 c.c. and 1000 c.c. of intravenous electrolyte and glucose, when his pulse is preferably below 120 and his systolic blood pressure is at least 100. If it cannot be brought above 90, there is indeed a grave risk in operating, though, at times, we must operate with a systolic blood pressure of 90, realizing that operation is the patient's only hope.

A haemoglobin determination is also taken, for a blood transfusion may be necessary. We also appreciate the hematocrit readings, since, not only fluids to combat dehydration, but a blood-volume-expander including plasma, may, on account of dehydration and blood concentration be more necessary than a blood transfusion (Elman, 1950).

A pre-operative roentgenogram is helpful and often gives a striking step-ladder effect, especially in cases of volvulus of the small intestine. It is especially helpful when one suspects volvulus of the sigmoid colon. The x-ray appearance of the distended large bowel, with the patient in the erect position, is usually distinctive. In such cases the volvulus may possibly be reduced by a barium colon enema. Or, when it is obvious that there is no strangulation, the patient may be placed in the knee chest position and a sigmoidscope passed in the usual manner with a gentle ballooning of the gut by air pressure. The lumen must always be clearly in view of the advancing sigmoidoscope. Success in the use of this method is unfortunately attended by a sudden rush of faeces toward the operator. But there is also the gratifying and rapid deflation of the abdomen. However, when the patient's condition is precarious or strangulation is suspected, and taking of the x-ray film might use up valuable time in the preoperative preparation, we feel it is better to omit it. Usually we must open the abdomen anyhow and the maximum point of tenderness is usually a good indicator of where the incision should be made. Usually also, the exact nature of the cause of obstruction cannot be determined by any means other than by laparotomy, with inspection and palpation of the abdominal contents. At that time one chooses the appropriate method of treatment, which need not be detailed here. Two facts only might be stated: It is always gratifying to find an uncomplicated volvulus and to untwist it. Also after the obstruction, whatever the cause, is relieved, and a rectal tube is inserted and intestinal contents flow out on the operating table, the ordinarily repugnant odour of faeces is most welcome. Where the peritoneal cavity has been soiled, it is most helpful, after aspirating the pus or faecal matter, to insert 1 gram of streptomycin and 4 to 5 lacs of penicillin, diluted with 20-30 c.c. of saline or distilled water. This is done through a catheter before closing the final stitch in the peritoneum. In such cases also we drain the abdomen by one or more cigarette drains.

To conclude, the factors in lessening mortality and morbidity appear to be:

1. Restoring fluid and electrolyte balance.
2. Decompression of the intestinal tract above the obstruction.
3. Blood transfusion or blood-volume expanders as may be necessary as determined by haemoglobin and hematocrit readings.
4. Early operation.
5. Penicillin, streptomycin and other antibiotics, parenterally and also into the peritoneal cavity when advisable.

We feel that spinal anaesthesia approaches the ideal form of anesthesia for these cases.

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THE INCIDENCE AND NATURE OF CONGENITAL CARDIAC ANOMALY IN MONGOLS

With a Case Report of Fallot's Tetralogy in A Mongol

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The frequent association of congenital cardiac malformations in Mongols is well recognised, and the figure of incidence has been cited by different authors from time to time. Thompson (1920) found heart lesions in 20 per cent of Mongolian idiots; Thursfield and Scott (1913) found heart disease in 16 per cent, Spuhler (1929) in 25 per cent, Cassel (1917) in 12·7 per cent and Edelhaus (1932) in 21 per cent.

Evans (1950) collected from the Great Ormond Street Hospital autopsy records of 28 congenital cardiacs in 63 Mongols—an incidence of 44·8 per cent.

Single or more case reports of Mongols with congenital cardiac anomaly have been cited by various authors. To mention a few: Gibson and Clifton (1938), Schleussing (1925), Gunn and Dickmann (1927), Robson (1931), Meecker *et al* (1935), Robinson (1941), Lightner (1939). Of these authors Robinson (*loc. cit*) while reviewing literature of the cases who had persistent atrioventricular communis as the congenital cardiac anomaly, found a total of 39 cases with that anomaly and of these more than 50 per cent were Mongols. Robinson's review included cases reported earlier by Morgan and Sprenkel (1936), Wolf and Levinson (1939), Mannheimer (1949), and Leech (1935). Leech (*loc. cit.*) reported 6 cases with congenital cardiac anomaly at the John Hopkins. Silvy (1934) reported 55 cases of Mongols with congenital cardiac anomally.

Clinically the congenital cardiac malformations in children in general is significantly lower than those noted in Mongols. Brown (1950) collected the data of the incidence from the figures quoted by White (1931). It appears that between 1-2 per cent of the cases of organic heart disease are of congenital origin. It is now widely expressed that with the continued advances in the methods of the diagnosis of the heart diseases the percentage might be slightly higher.

The nature of cardiac anomaly in Mongols—The reported anomalies have been widely different and as to the commonest nature of the anomalies in the Mongols, opinion is sharply divided. Potter (1952) while commenting on the nature of the cardiac anomalies in Mongolian idiots notes that 'although various malformations have been described the far more commonly observed than any other is a persistent atrioventricular ostium'. This malformation in her opinion is limited exclusively to the Mongolian idiots and has been designated popularly by various authors as the 'Mongolian heart'.

Abbott (1927) observes that 'the commonest cardiac anomaly in Mongolian idiocy is a defect of the interauricular septum, especially ostium primum'. But she actually wrote that in Mongols the defect 'in our experience is not infrequently of this type' (Abbott, 1924).

Robson (*loc. cit.*) reported a case with persistent atrioventricular communis associated with a defect in the base of the interventricular septum and ostium primum.

Meecker and Goetsch (quoted by Yannet, 1950) describe independently examples of cases with persistent atrioventricular communis. Gunn and Dickmann (*loc. cit.*) report three cases with congenital cardiac anomalies; one had defect of atrial septum with numerous perforations in the atrial septum, patent foramen ovale, defect of the upper part of the ventricular septum, persistence of a common atrioventricular ostium and abnormal development of the atrioventricular valves with extensive hypertrophy and dilatation of the heart; another with atrial septal defect with patent foramen ovale, defect of the upper part of the ventricular septum, common atrioventricular valves and patent ductus arteriosus and the third had the following lesions: common atrium and completely divided ventricles and imperfectly formed tricuspid valves—cor triloculare biventriculosum. Of the 7 cases of Gibson and Clifton (*loc. cit.*) the following lesions were present: persistent ostium primum with defect of the interventricular septum—one case, Fallot's tetralogy—two cases, ventricular septal defect—one case and patent ductus arteriosus—two cases.

Robinson (*loc. cit.*) while reviewing literature on the reports of the cases with persistent atrioventricular ostium states, "the total of cases reported to date is 39 and over half of these cases had mongolian features." He added to this series his observed case of persistent atrioventricular ostium in a Mongol.

Silvy (*loc. cit.*) notes maladie de Roger in 61 per cent of 55 Mongols with heart disease.

From the above discussion one might easily get the impression that persistent atrioventricular com-

munis is the usual or even the characterstic cardiac anomaly of the Mongols, inspite of the description of cases with other types of congenital cardiac anomalies by Levinson and Wolf (*loc. cit.*), Mannheimer (*loc. cit.*), Silvy (*loc. cit.*) etc.

Yannet (1950) states that the interventricular septal defect is the commonest cardiac lesion in Mongols.

Leech (*loc. cit*) described 6 cases with congenital cardiac anomalies in Mongols. Only one had common atrioventricular communis but also had patent foramen ovale and anomalies of great vessels in addition.

The nature of the cardiac anomalies in the 28 cases reported by Evans (*loc. cit.*) is shown in Table 1.

TABLE 1—CARDIAC ABNORMALITIES FOUND IN 28 MONGOLS

	Without patent ductus arteriosus	With patient ductus arteriosus
Large atrial septal defect, type uncertain	5	—
Persistent ostium primum ...	1	—
Persistent ostium primum and secundum	—	1
Absent auricular septum ..	1	—
Persistent atrioventricularis communis	3	1
Large atrial septal defect and ventricular septal defect (membranous)	1	2
Ventricular septal defect (membranous 6, more extensive 1) ...	6	1
Patent ductus arteriosus ..	—	1
Pulmonary infundibular stenosis	1	—
Pulmonary artery stenosis, atrial septal defect	—	1
Fallot's tetralogy (1 with atrial septal defect)	2	—
Aberrant right subclavian artery	—	1

Surprisingly enough the so-called Mongolian cardiopathy—persistent atrioventricular communis was detected only in 4 cases i.e. 14·2 per cent. On the evidence produced by these cases and the cases reported by Leech (*loc. cit.*) persistent atrioventricular communis does not appear to be the typical congenital cardiac anomaly in Mongols for it was detected only in 5 cases of total 34, much lower than 20 per cent. In Evan's (*loc. cit.*) opinion 'application of law of probability' of Taussig (1947) leads to the conclusion that 'if a Mongolian idiot has a congenital malformation of the heart which superficially resembles a ventricular septal defect and there is no cyanosis, the probability is that he actually has ventricular septal defect although he may have less common persistent atrioventricular communis."

It will not be out of place to compare the nature of the anomalies in Mongolian cardiacs with anatomical anomalies of the congenital lesions in children in general. It will, on the other hand, add a flavour of embryological importance and might throw light from the precise nature and time of arrest processes (as is thought to be the cause of congenital cardiac anomaly in Mongols) leading to the malformations of the heart, on the factors which are responsible for the production of the subject itself—Mongolian idiot.

Evans (*loc. cit.*) compared the anomalies of the Mongolian cardiacs with that of the 50 non-Mongol children having congenital cardiac lesions autopsied in the same period in the Great Ormond Street Hospital. Among the individual lesions in the latter group it is noted that persistent atrioventricular communis was absent, thus it appears that this lesion is less rare in Mongols than in the non-Mongols. Auricular septal defect in general is remarkably common in Mongols. Ventricular septal defects and patent ductus arteriosus are not so common as in non-Mongols. On the other hand, truncus arteriosus, transposition of great vessels and coarctation of aorta are rather unusual in Mongols (excluding the case of transposition of great vessels reported by Mannheimer (*loc. cit.*)

The most clear-cut difference between the lesions in the two series is the absence of defects due to malformations of the bulbus cordis (except Fallot's tetralogy) in the Mongols and of the common atrioventricular ostium in the non-Mongols.

Table 2 compares the individual anomalies of the two series according to the frequency rate Evans (*loc. cit.*).

'The common atrioventricular canal is divided and the ostium primum is closed by about 41st day of intrauterine life, the interventricular foramen closes by about 47th day (Hamilton *et al*, 1949), the second torsion of the bulbus occurs between the fifth and the eighth weeks (Brown, 1950) so that exaggerated torsion may occur after the 50th day ; the ductus arteriosus closes after the birth' (Evans, 1950).

While the cardiac anomalies are analysed in Mongols with the above stated landmarks of the embryology of heart it would appear that the basic developmental defects in Mongols occur very early in foetal life.

The case described here is a Mongol with Fallot's tetralogy.

TABLE 2—INDIVIDUAL ANOMALIES OCCURRING 2 OR MORE TIMES IN THE HEARTS OF 28 MONGOLS, OR 4 OR MORE TIMES IN 50 PATIENTS WITH CONGENITAL HEART DISEASE WHO WERE NOT MONGOLS.

Lesion	Mongol		Non-Mongol	
	No.	%	No.	%
Auricular septum, all defects	17	60	18	36
Auricular septum, ostium primum or secundum without persistent atrioventricular communis	12	43	15	30
Persistent atrioventricular communis	4	14	0	0
Ventricular septum—all defects	17	60	26	52
Ventricular septum, absent or nearly so ...	1	4	5	10
Ventricular septum, membranous defect without persistent atrioventricular communis ...	12	43	21	42
Patent ductus arteriosus ...	8	29	20	40
Truncus arteriosus ...	0	0	4	8
Pulmonary stenosis ...	4	14	3	6
Coarctation of aorta ...	0	0	4	8
Transposition of aorta and pulmonary artery ...	0	0	7	14

CASE REPORT

S, a male, aged 5 years was admitted on 8-1-53 in the paediatrics wing of the Medical College Hospitals, Calcutta, for diarrhoea. Past history revealed nothing significant. He is the fifth child of the sibsib of 7 children of which others are more or less healthy. Mother was 30 and the father 37 at the time of the birth of the baby. There was no history of eruptive fevers or any uterine troubles, e.g., gestational haemorrhage or threatened abortion during the pregnancy. Parents were apparently healthy.

The baby recovered from the attack of diarrhoea within 5 days after admission into the hospital.

On examination he appeared to be inert, apathetic to his surroundings, preferred to remain undisturbed. He passed his time constantly in an aimless desultory manner and did not grumble for anything. Growth of the baby was remarkably retarded and the body weight on admission was 16 lb. 7 oz. Facies had obvious mongoloid features and the head was brachycephalic. The nasal bridge was flat, the tongue big and fissured and relatively bigger than the small mouth cavity. The teeth were abnormally aligned. Dyspnoea was a prominent feature and present even at rest. Cyanosis was very marked and history revealed that the baby was 'blue' since birth. The baby preferred to remain in the squatting posture whole day and even slept in this posture at times. He had not learned to stand and walk or speak. The hands were 'podgy' with short and squant fingers. The little fingers of both hands were incurved and did not reach the last interphalangeal joint of the ring fingers. The skin of the hands was remarkably coarse with prominent transverse wrinkles on the palm. The classical 'penrose triangles' were noted in both palms. A big cleft was present between the great toe and the other toes of the feet. Clubbing of the fingers and of the toes were very marked.

The apex beat was in the 4th left intercostal space on the midclavicular line, slapping in nature. A systolic thrill was felt at the base of the heart along the left border of sternum and a harsh systolic murmur in the same position as that of the thrill was noted; which was conducted towards the mitral area but not transmitted towards the vessels of the neck. It was heard best in the recumbent posture. Pulmonary second sound was weak and the aortic second sound was loud. X-ray (Fig. 1, *vide* Plate) showed Coeur en sabot, no fullness in the region of the pulmonary conus, the upper portion of the left border of the cardiac silhouette in contrast to the normal convexity was concave, great vessels shadow narrow and the aortic shadow normal. L.O.A. view (Fig. 2): Right ventricle enlarged, aorta far forward from the spine and the pulmonary windows abnormally clear. R.O.A. view: left auricular enlargement and pulmonary conus fullness are not noted. Lung fields were abnormally clear oligaemic. The hilar shadow was increased but no expansile pulsations were visible on screening. The electrocardiogram showed: Right axis deviation with a heart rate of 96 per minute and sinus arrhythmia. Right ventricular hypertrophy and auricular enlargement.

Liver was 3 fingers enlarged, soft and not tender and spleen was 2½ fingers, soft and not tender.

The findings of the general examination and the cardiovascular system indicated strongly that it was a case of mongolism with Fallot's tetralogy. The patient died in the hospital following an attack of high fever and haemorrhage from the different sites probably of subacute bacterial endocarditis. No post-mortem was done.

SUMMARY

Incidence of cardiac anomalies in Mongols has been cited and the same has been compared with that of the children in general. Both clinical and post-mortem data have been cited. The nature of the congenital anomalies has been analysed and compared in both groups and the commonest lesion in mongolism has been put forward. A case with Fallot's tetralogy in the Mongol has been described as this lesion is very rarely noted in them in the literature.

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REFRESHER COURSE

CHRONIC COR PULMONALE

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The term cor pulmonale is not so familiar to every general practitioner as other types of heart disease are.

The heart and the lungs are organs which are very intimately connected. The lungs stand as a link between the right and left sides of the heart and as such, they should be physiologically considered part and parcel of the cardiovascular apparatus. Those cardiac disorders which result from the diseases of the lungs or pulmonary vessels are known as cor pulmonale or pulmonary heart disease. In chronic cor pulmonale, the main changes in the heart are enlargement of the right ventricle with or without failure due to some chronic disease of the lungs or pulmonary vessels.

The lesions in the lungs or pulmonary vessels must be fairly extensive before they can affect the heart.

The causes of chronic cor pulmonale are many. Whatever may be the aetiology, one factor exists in every case of chronic cor pulmonale namely, the presence of some degree of pulmonary hypertension. Before going into the causes, it will not be out of place, therefore, to discuss briefly the physiology of pulmonary circulation and the factors that may influence the pulmonary arterial pressure. The normal pulmonary arterial pressure is about S/D—22/8 mm. of Hg. The pulmonary capillary pressure is 5 mm. of Hg. The normal rate of pulmonary blood flow is about 5 litres per minute.

There are four factors which may increase the pulmonary arterial pressure, pulmonary vascular bed, rate of pulmonary blood flow, alveolar oxygen tension and left atrial pressure.

If there is reduction of or obstruction to the total cross section of pulmonary vascular bed, then there may be pulmonary hypertension. But the reduction or obstruction must be of a severe degree —at least two-thirds of the pulmonary vascular bed must be obstructed or reduced before, it can by itself cause any increase of pulmonary arterial pressure at rest. When one main pulmonary artery is ligated as during pneumonectomy there is no rise of pulmonary arterial pressure.

Pulmonary blood pressure does not rise in normal subjects unless the rate of pulmonary blood flow is increased about three-fold. Lowering of oxygen tension in the alveoli causes reflex pulmonary hypertension by vasoconstriction.

When pressure in the left atrium rises as a result of mitral stenosis or left ventricular failure, there is passive rise of pulmonary arterial pressure.

Of the four factors mentioned above, changes in left atrial pressure do not play any part in causing pulmonary hypertension in chronic cor pulmonale, but the other three, namely, reduction of or obstruction to the pulmonary vascular bed, increased rate of pulmonary blood flow and lowering of alveolar oxygen tension either singly or in combination play a very important part in

causing pulmonary hypertension and right heart strain in chronic cor pulmonale.

The causes of chronic cor pulmonale can be classified on an anatomical basis:

1. Alterations of Pulmonary Vessels:
   - (a) Main pulmonary arteries
     - i. Pressure of aortic aneurysm, or a new growth.
     - ii. Gumma.
   - (b) Pulmonary arterioles
     - i. Primary—spasm, arteriosclerosis.
     - ii. Obliteration by periarteritis nodosa, bilharzia ova, recurrent carcinomatous and other emboli, Buerger's disease etc.
2. Alterations of Lung Parenchyma:
   - (a) Secondary emphysema
     - i. Chronic bronchitis } Commonest
     - ii. Bronchial asthma } cause.
     - iii. Pneumoconiosis
     - iv. Chronic fibroid phthisis
     - v. Bronchiectasis
     - vi. Miscellaneous
   - (b) Primary emphysema.
3. Alterations of Thoracic Cage:
   - (a) Kyphoscoliosis
   - (b) Thoracoplasty.

Of the above causes, diseases of the pulmonary vessels produce chronic cor pulmonale by producing progressive severe pulmonary hypertension and the lung parenchyma is normal in these cases. Clinically, they may be called pulmonary hypertensive heart disease. In diseases of the lung parenchyma, the pulmonary vessels are seldom grossly affected, and ordinarily pulmonary hypertension is not severe. In these cases, anoxia plays a very important part in producing pulmonary hypertension which is usually slight or moderate. But precipitation of acute anoxia may raise the pulmonary pressure to a degree which may be severe enough to produce right ventricular failure. As anoxia plays the predominant part in this type of chronic cor pulmonale, it may be clinically called pulmonary anoxic heart disease. In kyphoscoliosis and thoracoplasty, the main change in the lungs is emphysema and the cardiac changes are secondary to it.

Pulmonary Hypertensive Heart Disease

As already mentioned, diseases of pulmonary vessels producing progressive, severe pulmonary hypertension and finally pure right-sided congestive cardiac failure are the characteristics of this type of heart disease. In some cases the aetiology of the severe pulmonary hypertension is not due to any known pathological conditions and these are called idiopathic or primary pulmonary hypertension. As these cases produce classical uncomplicated examples of pulmonary hypertensive heart disease, they require special mention.

Cause—The actual cause is not known. It is more common in women, and though it may occur at all ages, the common age incidence is between 20 to 30 years.

Pathology—The pathological changes are similar to that of systemic hypertensive heart disease, but here instead of affection of the left ventricle and systemic vessels, the right ventricle and the pulmonary vessels are affected. There is intimal thickening of pulmonary arterioles and small arteries with atheromatous changes in the big pulmonary arteries. The right ventricle is hypertrophied and dilated and when failure sets in the right atrium is also enlarged. Such changes are the result of continued severe pulmonary hypertension as in some cases there is very little abnormal in the pulmonary vessels, just as we may see healthy systemic arteries in some cases of systemic hypertension. The pulmonary hypertension is probably due to spasm of the pulmonary arterioles, the cause of which is not yet known. The lung parenchyma is healthy and so are the left ventricle and the left atrium.

Pathological physiology—The lung volume is normal and so is the vital capacity. The pulmonary arterial pressure is much increased and may exceed the systemic pressure on exertion. The pulmonary capillary pressure is normal, but the arteriolar resistance is much increased. The cardiac output is low and is maximum at rest. With exercise the output is not increased. The arterial oxygen tension is not diminished unless congestive failure is advanced, when it may drop to 80 to 90 per cent. The arteriovenous oxygen difference is increased.

Symptoms—Symptoms do not appear unless the disease is fairly advanced. Dyspnoea, palpitation, fatigue, anginal pain and syncope are the chief symptoms which at first occur only on exertion, the patient being comfortable at rest. Gradually the patient is uncomfortable even at rest, but orthopnoea, paroxysmal dyspnoea and haemoptysis do not occur.

Signs—Certain signs appear due mostly to the low cardiac output. The patient is cyanosed, the cyanosis being of the peripheral type. Extremities are cold, pulse small, blood pressure low, pulse pressure small, conspicuous 'a' waves in neck veins are characteristic.

The cardiac impulse is diffuse. Systolic lifting of the 3rd left intercostal space is visible and palpable and the pulsations of the pulmonary artery may also be seen and felt in the 2nd left intercostal space. The forcible closure of the pulmonary valves may be felt as a diastolic thud. There is no thrill anywhere in the precordium. The pulmonary 2nd sound is high-pitched. A presystolic gallop is heard in the epigastrium below the xiphisternum or in the 4th intercostal space just to the left of the sternum.

The rhythm of the heart is usually normal, rarely auricular fibrillation or flutter may occur in the late stages. The lungs are dry and show neither emphysema or any other abnomality. The liver is enlarged tender and pulsatile.

E. C. G. shows tall but not much wide P wave (P pulmonale) and evidence of right ventricular hypertrophy and strain.

X-ray shows small aorta, enlarged pulmonary arteries and enlargement of right ventricle and right atrium. The left atrium and left ventricle are normal and the peripheral lung fields are not congested.

Prognosis—The disease is progressive and ultimately pure right-sided failure with low cardiac output appears. Death usually occurs within two years from the onset of symptoms.

Treatment—Treatment is unsatisfactory. Oxygen inhalation is of no help. The usual drugs and measures employed for congestive cardiac failure are not very helpful when failure appears. Benrylindazoline hydrochloride and hexamethonium group of drugs may be of some help in lowering the pulmonary arterial pressure. Antithyroid drugs may help in prolonging life.

Pulmonary Anoxic Heart Disease

Causes—As already mentioned, emphysema secondary to chronic bronchitis with or without asthma is the commonest cause of pulmonary anoxic heart diseases ; in fact this is the commonest cause of chronic cor pulmonale.

Incidence—It is five times more common in men and 75 per cent of cases are over 50 years of age. Five to 10 per cent of all organic heart diseases are due to pulmonary anoxic heart.

Pathology—The lungs show changes characteristic of chronic bilateral hypertrophic emphysema with or without fibrosis, bronchiectasis or any other conditions which are responsible for producing the emphysema. The big pulmonary arteries show atheroma and the arterioles and small arteries intimal thickening. Some pulmonary capillaries are obliterated. The right ventricle is enlarged and so also is the right atrium.

Pathological physiology—The vital capacity is reduced to 1·5 litres or below. The arterial oxygen tension is lowered to about 60 to 80 per cent and there is retention of CO_2 in blood due to deficiency of gaseous exchanges in the alveoli. Breathlessness is partly due to gaseous abnormalities. The minute volume of respiration is increased and the cardiac output is enhanced to about 5 to 8 litres per minute. Some polycythaemia may occur.

In mild emphysema, there is obliteration of a few pulmonary capillaries which is not sufficient enough to cause any rise of pulmonary arterial pressure at rest or on exercise. There is also slight disturbance in gaseous exchanges in the alveoli which is compensated by increasing the minute volume of respiration. With further progress of the disease there is obliteration of more pulmonary capillaries and further lowering of alveolar oxygen tension leading to reduced arterial oxygen saturation. This anoxia tends to cause reflex pulmonary hypertension by vasoconstriction which is evidenced at first only on exercise. Further compensatory mechanisms come into play, the rate of pulmonary blood flow is increased by an increase of cardiac output and polycythaemia though not significant may occur. Under such circumstances, there are several factors which may cause pulmonary hypertension—reduction of pulmonary capillaries, anoxia, and increased pulmonary blood flow. Any one of these by itself is not severe enough to cause pulmonary hypertension, but all of them together may be sufficient enough to cause slight pulmonary hypertension at rest, and consequent enlargement of the right ventricle. When over and above, an acute infection such as acute bronchitis or bronchopneumonia supervenes or an attack of severe bronchospasm occurs there is precipitation of acute anoxia which causes reflexly severe pulmonary hypertension and the right ventricle may fail and congestive failure appears. With adequate treatment of the infection or bronchospasm, the anoxia may lessen, pulmonary pressure may be lowered and congestive failure may disappear.

Symptoms—The patient usually a male over 40 years, complains of cough or asthma for many years, and dyspnoea on accustomed exertion which is gradually progressive for 1 to 2 years. He may suffer from attacks of tightness in the chest with dyspnoea but not of typical paroxysmal cardiac dyspnoea or may complain of vague substernal discomfort but not of typical effort angina or he may consult his physician for oedema of legs which has developed recently.

Signs—The patient may feel comfortable in propped up posture, but may not object to remain

flat. In fact, some may prefer a recumbent posture as they feel more at ease in lying flat. This is due to the fact that in recumbent posture, the diaphragm is pushed up and so in a case of emphysema, respiration is more easy as the diaphragm gets a better chance to act. In other types of congestive failure, a propped-up posture is nearly always preferred to that of recumbency.

General survey reveals that the patient is cyanotic, the cyanosis being of the central type. The neck veins are full during expiration, the extremities are warm, digital throbbing and capillary pulsation may be present. There is some tachycardia ; clubbing of fingers may be present.

Systemic examination reveals that the lungs are emphysematous. The heart is camouflaged by the expanded lungs. Cardiac impulse is usually invisible and impalpable ; but epigastric pulsation may be seen. Cardiac dullness is obliterated. Heart sounds are feebly heard, the pulmonary second sound is feeble or inaudible. A right-sided presystolic gallop to the left of the sternum in the 4th space or below the xiphisternum may be heard. There is no murmur, apparently it is a quiet heart. The liver may be just palpable, but not tender and the hepatojugular reflux is negative.

When congestive failure appears due to acute infection or bronchospasm, neck veins become congested during expiration and inspiration, oedema of the feet appears and liver is more enlarged and tender. The hepatojugular reflux is positive.

Skiagrams show enlarged pulmonary arteries and enlargement of right ventricle and right atrium. Early enlargement of the pulmonary artery is best seen in the right anterior oblique view. In more advanced cases a fullness or convexity of the pulmonary bay may be seen in P.A. view with dilatation of the main branches of the pulmonary artery. Enlargement of the right ventricle may not be detected in P.A. view unless it is gross, due to the low position of the diaphragm and expanded condition of the lungs which make the cardiac shadow appear normal or even smaller. The lungs will show emphysema and associated pathological changes.

E. C. G. will show pulmonale type of P wave and evidence of right ventricular hypertrophy and strain.

Prognosis—Prognosis of pulmonary anoxic heart disease is bad. Once congestive failure develops, a fatal outcome is usually expected within 1 to 2 years. Repeated attacks of congestive failure as seen in other types of heart diseases are not common.

TABLE I

	Emphysema without cor pulmonale	Emphysema with cor pulmonale ; no failure	Emphysema with cor pulmonale ; slight failure	Emphysema with cor pulmonale ; severe failure
Congested neck veins	+ on expiration only	+ both on inspiration and expiration	++ both on inspiration and expiration	+++ both on inspiration and expiration
Pulse pressure ...	Normal	Large, slight water-hammer type	Large	Small
Liver	Just palpable, not tender	Enlarged +, not tender	Enlarged ++, tender	Enlarged +++, tender
Hepatojugular reflux	Negative	Negative	Positive	Positive
Oedema	Nil	Nil	Slight	++
Venous pressure ...	Normal	High, normal or slightly raised	Definitely raised	Definitely prolonged
Arm-lung circulation time.	Normal	Normal	Slightly prolonged	Definitely prolonged
Digitalis etc. ...	Contraindicated	Contraindicated	To be given cautiously, effect watched	Definitely indicated

Treatment—As already discussed, precipitation of congestive failure in pulmonary anoxic heart disease occurs due to supervention of acute bronchopulmonary infections or of spasm. Adequate treatment of such infections or bronchospasm will delay the onset of congestive failure.

When congestive failure has developed, here too, treatment of acute infection with penicillin or other antibiotics and chemicals, and treatment of bronchospasm with adrenaline, noradrenaline, aminophylline and antihistaminics as necessary should be considered as the most important step for managing the failure. Morphine is dangerous in this condition and should never be given, if necessary, pethidine may be given instead. Oxygen therapy is beneficial, but continuous oxygen administration may be dangerous. In pulmonary anoxic heart disease, there is retention of CO_2 in blood along with anoxia. The respiratory centre becomes rather hyposensitive to CO_2 in blood along with anoxia. Respiration is partly carried out due to the stimulus of hypoxia. If oxygen is administered continuously, the arterial oxygen tension will be restored to normal and the stimulus to respiration will be taken off. Respiration will be shallow and further retention of CO_2 in blood will produce CO_2 narcosis; the patient feels a sense of fullness in the head, he is disoriented, there is sweating, myoclonic movements appear, and ultimately he becomes unconscious though O_2 tension in arterial blood is over 90 per cent. This danger of continuous oxygen therapy should be remembered and oxygen should be administered intermittently.

For management of congestive failure the usual drugs (digitalis, diuretics) and measures (salt restriction, venesection) are indicated though their effect is far less satisfactory in pulmonary anoxic heart disease. We should remember that in this type of heart disease, up to a certain level, a raised venous pressure is beneficial and compensatory and maintains a high cardiac output which is necessary under such circumstances. The usual drugs and measures employed in congestive failure reduce venous pressure, and if they are administered injudiciously in pulmonary anoxic heart disease, they may lower the venous pressure to a level which instead of improving the circulation may actually make it worse. It is not always easy to decide at what stage of pulmonary anoxic heart disease, they should be adopted and no hard and fast rules can be laid down. Each case should be judged on its own merits. Table 1, however, may be of some help to the general practitioner.

The picture of anoxic pulmonary heart disease depicted above stands true for the majority of cases of emphysema heart. But there are a small group of cases which shows a different picture. In them, the predominant pathology is progressively severe pulmonary hypertension ultimately leading to congestive cardiac failure without any superimposition of acute infection or bronchospasm. They show a picture with low cardiac output, mixed type of cyanosis, cold extremities, small pulse, low pulse pressure and giant venous 'a' waves. X-ray changes are progressive and permanent and E. C. G. shows marked P pulmonale and right ventricular hypertrophy and strain. Within 1 to 2 years they end fatally with the development of congestive failure of the low output type. They resemble pulmonary hypertensive heart disease more than anoxic heart disease. It is difficult to say whether there are two types of pulmonary anoxic heart disease, one with high cardiac output and slight pulmonary hypertension which becomes severe only during acute infection or bronchospasm, and the other with gradual, progressive, severe, pulmonary hypertension with low cardiac output, or some cases of the first group due to some reasons ultimately pass into the second group or there are certain individuals who due to some physiological and anatomical peculiarities in their pulmonary circulation react differently in response to emphysema.

TREATMENT OF SOME COMMON COMPLICATIONS IN ACUTE FEVERS

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During a febrile disturbance, events sometimes move so swiftly that a complication may completely overwhelm a patient within a few hours and the physician may suddenly find himself at his wits' end to cope with the emergency. In a situation like this a physician who relies on his own clinical judgment and experience and makes balanced but bold decisions is likely to come to grips with the problem much more effectively than one who is inclined to be completely dependent on his laboratory colleagues.

In the present communication we shall endeavour to deal with the clinical assessment and management of some of the serious complications that commonly occur in acute fevers.

Hyperpyrexia

A patient is usually labelled to be suffering from hyperpyrexia when his body temperature rises above 105°F. (strictly 106°F.). In winter months a temperature of 105°F. may be tolerated but in summer months quick measures should be adopted once the temperature goes over 103°F. as there is always a risk of hyperpyrexia supervening in such cases. In several parts of India, temperature during summer may range between 110°-116°F., yet the body is able to maintain normal temperature by sweating, as evaporation of the sweat cools the body. In summer, a patient may start a febrile disturbance with some infection such as malarial fever, with the temperature rising very quickly to 105°F. or over. The sweating mechanism may get deranged and the patient may soon get into a state of hyperpyrexia. Whenever the body temperature reaches a level of 107°F. or over, one should regard it as a desperate emergency as the temperature has to be lowered within a few minutes if life has to be saved. Once the temperature remains at that level for a few hours, irreversible damage to the heat regulating centre may occur and the patient dies quickly of medullary paralysis. Hospitalisation is usually advocated under these circumstances. It is desirable that all our modern hospitals should have some air-conditioned rooms or small wards where cases of hyperpyrexia should be treated. Failing this a separate room should be selected where ice slabs are kept and a fan and "khas khas tatti" are provided. The best methods to lower the body temperature are to stimulate the natural conditions as far as possible. The temperature of the room in which the patient is being treated must be lowered. The next step should be to imitate the normal sweating mechanism of the body. The latter objective can be achieved in the following way. The patient should have all the clothes taken off and the body should be wrapped in a wet sheet. A big piece of mackintosh should be laid on the bed to prevent the soiling of bed linen. The bed of the patient should now be placed directly under the fan. The evaporation of water from the wet sheet with a fan moving above will reduce the body temperature. At the same time an ice cap should be applied to the head. An iced water enema may lower the temperature further. Some patients in the hyperpyrexial state are very delirious and restless. It is better to give 6-8 c.c. of paraldehyde intramuscularly to quieten such a patient. One should not loss sight of the fact that at such high levels of temperature the patient needs fluid badly. One should put such a patient on intragastric drip of plain iced-water provided the patient is not vomiting and is free from convulsions. Intravenous drip with glucose saline may be more useful but it carries the risk of a pyrogen reaction. It is a common experience in summer months that glucose saline, if not pyrogen free, may further aggravate the situation by a rigor followed by still higher levels of temperature. Intragastric and rectal drips may be more desirable under such circumstances. If pyrogen free glucose saline is available and all precautions necessary for intravenous transfusions can be taken, the intravenous route is the route of choice. The amount of fluid and speed of administration will vary from case to case depending on the state of dehydration.

Besides hyperpyrexia due to heat stroke, two of the common causes of hyperpyrexia are either an attack of malaria or some acute infection. Along with relevant laboratory examination, it should be a routine practice to give 2-5 lac units of penicillin intramuscularly and 10 grains of quinine bihydrochloride intravenously as an initial dose, pending laboratory reports. Quinine should preferably be given in the intravenous drip. Further specific treatment will depend on the underlying cause of the fever, but it may take time to discover that.

Peripheral Circulatory Failure

Peripheral circulatory failure is one of the most dreaded complications in an acute febrile state. The exact mechanism of this circulatory disturbance is not known. The pathological picture is the same as met with in a case of surgical shock but it may occur more gradually in fevers. Clinically there will be a rise in the pulse rate, a fall in the blood pressure and the patient may sometimes present an ashen appearance. In those fevers where dehydration is an added factor the patient may show a picture of oligaemic shock. Sometimes toxaemia, dehydration and peripheral circulatory failure may all be present in combination in the same patient. One must bear in mind that the chances of recovery depend on the early recognition of peripheral failure. When the pulse rate is over 140 per minute and systolic blood pressure has fallen below 90 mm. of Hg., peripheral failure is already established. The first principle in the treatment is to tackle the aetiological factor of a particular fever with effective chemotherapy. There are however certain precautions to be observed. In a case of falciparum malaria of pernicious type with peripheral circulatory failure it may be risky to give quinine intravenously till the blood pressure has been first raised by drip therapy and other restorative methods of

treatment. It is well known that quinine may further lower the blood pressure of a moribund case. So, meanwhile, other methods of specific treatment against malaria may be preferable in such cases. Similarly in a case of typhoid fever with peripheral circulatory failure one should not use heavy doses of chloramphenicol.

The second step in the treatment is to help and restore the circulatory dynamics to normal as quickly as possible. The patient should be given 5 per cent glucose saline drip. The amount and speed of administration will depend on the condition of the patient and associated dehydration. In an average case 4-6 litres may be given in 24 hours. In a patient who is bed-ridden for several weeks with fever, blood or plasma transfusion may give additional help. Other measures to be taken in these cases are the various means adopted to raise the blood pressure by stimulants. Nikethamide enjoys a good reputation. It is doubtful whether it helps very much unless given intravenously. In recent years more effective drugs have been introduced. Methyl amphetamine hydrochloride 15-20 mg. may be given intravenously in a rapid drip and can be repeated at 4-6 hours' interval intravenously or intramuscularly. More recently *l*-nor-adrenaline has come into use in peripheral circulatory failure. 2 to 3 mg. may be given in a running drip. The speed of the drip and dosage can be regulated according to the response and requirement of an individual case. In a case of associated anoxaemia oxygen administration may help the patient.

Dehydration

Dehydration is a common complication in fevers. It may be due to insufficient intake of fluids or due to loss of fluids and salts by vomiting and diarrhoea. It is now recognised that a patient may suffer from water dehydration, or salt dehydration, or both, i.e., mixed type where water and salts are both needed to restore the fluid and electrolyte balance of a patient.

Water dehydration would occur in a fever case when fluid administered is too small or when the patient is unable to take sufficient amount due to some other reason, e.g., a comatose or a delirious patient would not take required amounts of fluids. Sometime a patient may be too weak or exhausted to take sufficient fluids. If water depletion occurs, the fluid in the extracellular compartment becomes hypertonic in relation to the cellular compartment. Water is thus drained out of the cellular compartment to establish osmotic equilibrium. It is thus clear that water depletion causes mainly cellular dehydration. Urinary output falls to the minimum. Such a patient will complain of extreme thirst and marked dryness of the mouth. The tongue will be dry and coated. Salt depletion (secondary dehydration) occurs in fever cases when profuse sweating is going on or when marked diarrhoea and vomiting are present with great electrolyte loss. If such a case is given plenty of water but no salt there will be salt dehydration. In salt depletion the fluid in the extra-cellular compartment will be in a hypotonic state in relation to the cellular compartment. The kidneys try to maintain osmotic equilibrium by excreting water. The patient however presents a picture of peripheral circulatory failure and oligaemic shock. Such a case will complain of lassitude, apathy, mental confusion and stupor. Muscular weakness is well marked and later on actual cramps might occur. Anorexia and vomiting may lead to further salt loss and the patient may get into a vicious circle. The skin becomes inelastic ; the face is pinched. The eyeballs get sunken and cyanosis of the lips may occur. There is marked rise of pulse rate and fall in blood pressure. Two deceptive negative findings should be borne in mind. If the patient is taking enough water he will not complain of any thirst and no evidence of oliguria will occur. In terminal stages, however, anuria may supervene due to marked fall in blood pressure. Absence or diminution of chlorides in the urine is a very reliable finding in those cases.

In a large majority of patients, however, mixed dehydration occurs and the presenting clinical picture will be of both water and salt depletion. Such patients will complain of thirst and dryness of the mouth with associated features of oligaemic shock.

Treatment of dehydration—Maintenance of a fluid chart showing intake and output is an essential procedure in the management of a fever case. In hospitals this practice should be the rule. When treating a patient at home the attending physician should give detailed instructions about the fluid intake. The urinary output is a good guide to the fluid intake and should be watched. Similarly one should see that the daily intake of salt does not go below 10 g. In fact during summer months the requirements may be more. The patient should be given on the average 4-5 litres of fluids in 24 hours in winter months but in summer months at least 6-8 litres are required.

In case of water depletion, if the patient can take water by mouth it is ideal to use this route. In a comatose patient intragastric drip can be given consisting of plain water. Parenterally water can be given only as 5 per cent glucose solution. In a mild case total fluid requirements in 24 hours

may be 1·5 litres; in a moderately severe case 4 litres and in a very severe case 8-10 litres may be needed. The rate of administration will depend on the condition of the patient. In pure water depletion one is not in a desperate hurry to replace the body fluids. The amounts mentioned above can be given in 24 hours. If however salt depletion is also present one has to adopt immediate measures to save the patient from a quick catastrophe.

In case of salt depletion assessment of the case has to be made from the general clinical state, and urinary chlorides of the patient. The fluid to be administered will be isotonic saline and the amount will vary from 4-8 litres depending on the degree of dehydration.

The route of administration has to be intravenous in these cases as the patient cannot drink such large amounts of isotonic saline. When salt depletion is profound, one must quickly combat the oligaemic shock. The rate of administration should be 1 litre in the first hour and a half. This should be followed by 1 litre every 4 hours. When the B.P. has come to normal half a litre every four hours will suffice.

In mixed dehydration it is better to start with 5 per cent glucose in half strength normal saline first which can be reduced to ⅓rd normal saline later and still later only 5 per cent glucose is needed. It may not be out of place to emphasise the special importance of dehydration in children. In adults dehydration is a serious emergency but in children dehydration is to be regarded always as a grave disaster. Young infants stand dehydration very poorly. One has, therefore, to cope with the emergency without delay. In children one should start with 5 per cent dextrose in half strength or one third strength normal saline and later continue with 5 per cent dextrose in one-fifth strength physiological saline. Children do not need normal saline. The intravenous route should be used and it is the only reliable route for combatting dehydration. Half-hearted measures will not do. With a little practice dissection of the internal saphenous vein can be learnt easily. In infants during the first year of life the amount of fluid requirement in 24 hours has to be calculated according to the formula of 2½ ounces per lb. of the body weight plus the fluid lost in diarrhoea and vomiting.

Delirium

A patient in a state of delirium is a great cause of anxiety to the relatives, and in the wards such a case is always a headache to the nursing staff. The clinical picture varies from case to case. In a mild case there may be only some mental confusion and hallucinations may be present. On the other hand, a patient may become noisy, violent and abusive. The latter type of case may have to be restrained. It is advisable to keep such patients in rail-cots and strapping of the limbs may have to be resorted to. Feeding becomes a great problem and a patient has to be quietened before any effective nursing, feeding or therapy can be administered. In a mild case the usual chloral bromide mixture may be useful or sodium phenobarbitone grain 1½ to 3 may be given intramuscularly. Chloral hydras is an ideal drug for a delirious child.

In moderate and severe cases the best method to control delirium is to give 6-8 c.c. of paraldehyde intramuscularly and it can be repeated every 6 hours if required. In a violent case paraldehyde and sodium phenobarbitone may have to be combined. Hydrotherapy and restorative treatment should be applied at the same time. Morphine is contra-indicated. It may be stressed here that lumbar puncture may sometime prove very helpful. It relieves the intracranial tension and a delirious patient may be thus subdued when uncontrolled by other measures. Children, neurotic adults, and alcoholics are more prone to delirium. One must bear in mind the above factors before judging the gravity of an illness of a patient in a delirious state.

Convulsions

A convulsive fit in a febrile state is very common in the case of children. In adults convulsions usually occur in those fevers where either toxaemia is very marked or some cerebral complication like meningitis or encephalitis is present. Cerebral malaria as a cause of febrile convulsions is quite common in this country. Lastly they may occur in a case of heat stroke and hyperpyrexia. One should, however, bear in mind that repeated convulsions may themselves produce hyperpyrexia as is seen in a case of status epilepticus.

For convulsions in adults the ideal drug to use is paraldehyde intramuscularly; 6-8 c.c. can be given every 4-6 hours. It may be supplemented by sodium phenobarbitone by injection. The dose varies from grains 1½ to 3 depending on the severity of the case. In children chloral hydras is the drug of choice. An infant even in the neonatal period stands chloral very well. The dose will vary according to the age of the patient. An initial dose of 2-4 grains to a child of 1-2 years may be given. Subsequent doses may be smaller

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and they can be repeated 4-hourly. Children below 1 year should be given one-grain dose of chloral hydras.

Coma

Come is a very serious complication of fevers. Cerebral malaria and heat stroke are common causes in this country. Virus encephalitis may produce an overwhelming illness with high temperature, convulsions and coma. A comatose patient needs careful nursing. Treatment will depend on the underlying pathology. Hydrotherapy, chemotherapy, restorative treatment and rehydration therapy all may be needed in a case of coma. These have already been discussed in detail. Lumbar puncture is an essential procedure in every case of coma both from the point of view of diagnosis as well as of treatment.

Toxic Purpura

In many infectious diseases toxic purpura or haemorrhagic manifestations might occur and they usually mean a dangerous state. Various exanthemata, typhus fever, meningococcal infection and septicaemia are known to produce severe haemorrhagic states. A patient may bleed from the mucous membranes or he may present purpuric spots in the skin or may show both. The greatest danger lies in a patient having purpuric haemorrhages in the internal organs, particularly in the brain. In these conditions haemorrhages may occur due to damage to the capillary endothelium caused by toxaemia or there may be acute depression of the platelets. Such patients are gravely ill and the condition is called "purpura fulminans". In rare cases haemorrhages may occur in the suprarenals and the patient will present a picture of Waterhouse-Friederichsen syndrome. The patient presents a picture of peripheral circulatory failure with marked asthenia, prostration, vomiting, diarrhoea, abdominal pain and coma. Treatment of such cases is effective antibiotic therapy. In addition other heroic measures have to be adopted. For circulatory failure associated with purpura and in cases where there is acute depression of blood platelets blood transfusion must be given. Stimulant line of treatment with methedrin or noradrenalin is needed in addition. High doses of vitamin C are recommended. Recently some of these cases have been treated with high doses of cortisone. It is one of the gravest emergencies in fevers with hyperacute infection causing high mortality. Quick and emergent measures should be adopted as the patient may succumb within twenty-four hours.

SPECIAL ARTICLE

CURRENT CONCEPTS OF IRON METABOLISM WITH SPECIAL REFERENCE TO IRON TRANSPORT IN BLOOD

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Iron being an important element essential for haemopoiesis its excretion is minimal. It is achieved in the human body through the binding of circulating iron with protein. Another significant feature of iron metabolism is that the iron balance in the body is maintained by the regulation of absorption according to body needs rather than by excretion of unwanted excess. Absorption of iron is in turn regulated by the state of saturation of the storage depots like liver, spleen and bone marrow. It is due to these peculiarities of the requirements, absorption and conservation of this element that considerable attention is being paid to the various phases of its metabolism.

Requirements

Sufficient iron to balance the small daily excretion of this element is ordinarily derived from everyday foodstuffs. Dietary iron exists in inorganic and organic forms. The former passes readily into solution and gets absorbed while the latter chiefly in the form of porphyrin compounds has to be broken down before ionic iron is liberated. There is no accurate method to determine as to how much of the food iron can be converted into the ionic state and what proportion of the iron content can be rendered available for haemoglobin synthesis.

Absorption

Much is now known about iron absorption. Its various aspects have been well reviewed by Cartwright (1947) and Hynes (1948). It is now well established that the iron is absorbed from the stomach and the small intestine, chiefly the upper part. There is evidence that absorption can occur from colon. Radioactive iron has been of great help in understanding this phase of iron metabolism. Absorption of iron is complete in 4-8 hours because when radioactive iron is fed to dogs, it appears quickly in the plasma and reaches its peak in 4-8 hours. Numerous factors have been found to in-

fluence the absorption of iron. It has been seen that greater the dissociation of iron salts the greater will be the absorption. Thus the dissociation of iron salts is greater in acid solutions and slight above pH 5. This is the reason why the absorption of iron diminishes beyond the duodenum. Again substances which form insoluble salts with iron diminish its absorption. Examples being phosphates (Brock and Taylor, 1934) and phytic acid (McCance *et al*, 1943) which reduce the absorption of iron. Moore *et al* (1939) studied the absorption of iron after oral administration by estimating the level of plasma iron at various intervals and concluded that ferrous salts are better absorbed than ferric salts. The relation between bodily needs and iron absorption is very interesting. Hahn *et al* (1938) showed that in chronic haemorrhagic anaemias in dogs, absorption of radioactive iron was greater than in normal dogs. Balfour *et al* (1942) found greater absorption of radioactive iron in iron-deficiency states. Hynes (1949) showed that iron absorption is increased when body stores are depleted by repeated bleeding and thus the iron reserve of the body exhausted. Jasinski (1950) determined the extent to which iron absorption is disturbed by the presence of infection and malignant neoplasms. In pure infective anaemias he found that the serum iron level is not much affected by oral iron therapy. In other words the iron absorption is very little. Crawley (1952) performed a number of absorption tests in anaemias. In a normal subject he found a serum iron rise of 145μg./100 c.c. after the oral administration of 18 g. of ferrous sulphate. In iron deficiency anaemia it was 235 μg. and in pernicious anaemia 31 μg./100 c.c. serum. This showed that iron absorption is increased in iron deficiency states and decreased in pernicious anaemia. It should be made clear at this stage that the amount of circulating radioactive iron, i.e., radioactive iron present in circulating haemoglobin is not a reliable index of iron absorption. Dubach *et al* (1948) found that in untreated pernicious anaemia only very small amounts of radioactive iron appeared in the blood stream. After the injection of liver extract, however, the proportion of radioactive iron in circulating blood is considerably increased. This suggests that in untreated pernicious anaemia the iron was absorbed and stored somewhere else in the body and when later on reticulocytosis was established it was utilised for haemoglobin formation.

Mechanism for the Control of Iron Absorption

This has been studied in considerable detail by Hahn *et al* (1943 and 1943a). They found that oral administration of an iron salt a few hours before the administration of radioactive iron reduced the absorption of the latter. They thought it to be due to mucosal block produced by the iron salt given before. These authors also did not find any correlation between the concentration of iron in the gut and the plasma. In order to explain this they postulated the existence of an iron acceptor mechanism. This they said is present in the gastric and intestinal mucosa. In normal persons the acceptor mechanism becomes saturated in 1-2 hours. Desaturation takes several days. Granick (1946, 1946a, 1946b and 1947) suggested that a brown coloured protein, ferritin, is present in the mucosa of the intestine. This compound is formed by the union of a colourless protein named apoferritin with a particular kind of ferric hydroxide. The ferric hydroxide in ferritin is curious in that it is magnetically distinguishable from iron hydroxide found elsewhere in nature. Ferritin contains a variable amount of iron which may reach as high as 23 per cent. After feeding with iron an increase in ferritin is found in the gastrointestinal tract. In the duodenum ferritin content reached a peak in 7 hours and reached the normal levels again on the 6-7th day. These changes can be correlated with the course of saturation and desaturation of Hahn *et al* (1943 and 1943a). According to Granick's view the iron is absorbed by the mucosal cells in ferrous form. The receptor cells of the intestinal mucosa contain apoferritin. In the mucosal cells an equilibrium exists between the ferrous iron of the cells and the ferric iron of ferritin. In the presence of ferritin the cells are considered to be physiologically saturated with ferrous iron. It is only when ferrous iron has decreased to a point where the cells are no longer physiologically saturated with ferrous iron that more iron will be absorbed. Granick has shown that before the administration of iron there is very little ferritin in the gastric and intestinal mucosa and practically no apoferritin. Absorption of iron probably causes rapid formation of the specific protein to which it can become attached. Removal of iron from the ferritin causes apoferritin also to disappear. Thus in iron deficiency anaemias increased absorption of iron is due to fall in serum iron level which causes ferritin iron to pass into the blood stream. This produces a disturbance in the equilibrium in the ferrous iron in the cell and the ferric iron of ferritin. Some of the ferrous iron in the cell is shifted to form ferric iron of ferritin. Hence more of ferrous iron is absorbed by the cell to make up for the deficiency produced by the shift of ferrous iron of the cell. Thus the amount of iron absorbed is regulated by the amount of ferritin in the muco-

sal cells of the gastric and intestinal wall and this in turn is related to the need for iron in the body.

Transport

Moore *et al* (1939) showed that iron is absorbed from the alimentary tract into the blood stream and not into the thoracic duct. From then onwards plasma is the chief medium for the transport of iron to the storage depots and the sites for erythropoiesis. The fact that this iron is protein bound is now well established because it is nondialysable, does not appear in the ultrafiltrate unless acidified and is precipitated with the proteins. Holmberg and Laurell (1945) were able to study the capacity of the serum to bind iron *in vitro* by means of a colorimetric method in which dipyridyl was used. They found this saturation limit in 10 normal individuals to be 312 μg./100 c.c. serum. The values ranged from 264-366 μg. Laurell (1947) did some more work and pointed out that in a normal individual 300 μg. (roughly speaking) is the maximum that can be bound over by the iron binding protein. Since the serum iron is about 100 μg. per 100 c.c. serum, 200 μg. can still combine with the iron binding protein in 100 c.c. serum. In other words, normally one third of the protein is saturated with iron and the saturation percentage is 33 per cent. Both the values may vary in pathologic states. Laurell (1947) tried to find out whether the iron combined with this protein was in the ferric or ferrous state and could not come to any definite conclusion. Granick, has, however, assumed that iron exists in the plasma in the ferric form because of the high oxygen tension in the blood.

The protein fraction with which the iron is bound, has been variously named as iron binding protein, metal binding protein, transferrin and sideroglobin. Laurell (1951) objected to its being called metal binding protein on the grounds that this name is somewhat misleading, as copper *in vivo* is transported with another protein caeruloplasmin and not with the metal binding protein as was supposed earlier. He prefers to call it transferrin as he thinks that plasma is the real carrier of iron as haemoglobin is a carrier of oxygen. The nature of this iron-binding protein has also been thoroughly investigated. Schade and Caroline (1946) localised this iron-binding protein in fraction iv-7 of Cohn and his associates. He has shown that a progressive red colour develops on the addition of iron to this protein until it becomes saturated. At this point of saturation there is a break in the colour curve which corresponds to the exact point at which free iron can be demonstrated by bio-assay. Surgenor, Koeschelin and Strong (1949) described the spectrophotometric absorption curve of the iron protein combination. This forms a basis for the determination of iron binding capacity of serum. They also found that this fraction is a B_1 globulin with a molecular weight of 90,000. It constitutes about 3 per cent of the total plasma proteins. Its exact quantity has been found to be 0·27 g. per 100 c.c. of serum. Rath and Finch (1949) have determined that 1 mg. of this protein combines with 1·25 μg. of iron. Fiala and Burke (1949) discussed the mode of iron binding by this siderophyllin, conalbumin, hydro-oxylamines and aspergillic acid. They concluded that two atoms of iron are attached to one molecule of globulin.

The normal serum iron concentration has been determined by several workers in the past (Holmberg and Laurell, 1945; Cartwright and others, 1946; Laurell, 1947; Cartwright and others, 1948; Rath and Finch, 1949; Totterman, 1949; Anderson, 1950; Pirrie, 1952; Gitlow and Bayers, 1952; Davies, Levine and Oberholzer, 1952; Ramsay, 1953; Agarwal and Misra, 1954). Roughly speaking mean serum iron concentration is about 120 μg. per 100 c.c. serum.

Vahlquist (1941) reported a diurnal variation of serum iron in normal subjects during the day. 15 normal subjects had a mean serum iron level of 135 ± 10·6 μg./100 c.c. serum at 8 A.M. and by 6 P.M. a fall of 36·3± 9·2 μg./100 c.c. had occurred. Recently Patterson, Marrack and Wiggins (1953) have studied the diurnal variation of serum iron in various haemopoietic disorders. These authors found that the diurnal variation was diminished in aplastic anaemia, untreated pernicious anaemia, and iron-deficiency anaemia. Full treatment of the last two conditions restores a normal diurnal variation. Diurnal variation was also diminished in various conditions associated with increased erythropoiesis. Examples are polycythaemia vera, congenital haemolytic anaemia, nocturnal haemoglobinuria. It is suggested that decreased diurnal variation of serum iron level may be related to increased plasma iron turnover.

Pirrie (1952) pointed out that the level of serum iron decreased as the age advanced. Vahlquist (1950) discussed the cause of sexual differences in erythrocyte, haemoglobin and serum iron levels in human adults.

Iron is elevated in conditions of iron excess such as transfusion haemosiderosis, haemachromatosis, and in conditions where haemoglobin synthesis is disturbed as in pernicious anaemia, refractory anaemia and cirrhosis liver uncomplicated by blood loss. Cartwright *et al* (1948) found lower serum iron values in iron deficiency anaemia, haemorrhagic states and infection. Agarwal and Misra (1954) reported a fall in serum iron level in iron-deficiency

anaemias, anaemias of infection, malignant neoplasms and ankylostomiasis. Higher values have been reported in aplastic and haemolytic anaemias. Similar results have been reported by other workers also (Rath and Finch, 1949; Bendstrup, 1953). Pirrie (1952) studied the relationship between serum iron and copper in patients with neoplastic disease. He found serum iron levels to be lower while serum copper levels were significantly higher. Fredrickson (1953) discussed the significance of serum iron in the differential diagnosis between gastric carcinoma and peptic ulcer. In 13 patients with gastric cancer the average for serum iron was 64·2 μg./100 c.c. serum with a standard deviation of ±31·6. The averages for gastric ulcer (14 cases) are 145·5 μg. and for duodenal ulcer (24 cases) are 105 μg. per cent. This shows that there is about the same amount of serum iron in peptic ulcer cases as in normal cases. In bleeding ulcers, however, it is low. In cancer in general it is generally low even in cases with no real anaemia. Sideropenia is explained by supposing it to be a primary condition caused by the toxic influence or the iron consumption of the quickly growing tumours. In the case of gastric cancer a disturbance of absorption may play a part in producing sideropenia.

The total iron binding capacity and saturation percentage have also been determined by several workers in recent years (Laurell, 1947; Greenburg, *et al*, 1947; Cartwright and Wintrobe, 1949; Rath and Finch, 1949; Totterman, 1949; Bendstrup, 1950; Gitlow and Bayers, 1952; Agarwal and Misra, 1954b). The mean values have been found in the neighbourhood of 320 μg./100 c.c. serum. Cartwright and Wintrobe (1949) found a fall in the iron binding capacity in states of chronic infection and rise in iron deficiency anaemias. Rath and Finch (1949) report their observations in 30 normal subjects and 105 patients. They found a fall in the iron binding capacity in cancer, infection, liver and kidney disease. Bendstrup (1953) also estimated the serum iron and iron binding capacity in acute and chronic infections. He found a decrease in serum iron and iron binding capacity in 133 patients with chronic rheumatoid arthritis. It was also lowered in both the acute and chronic infections. Mukherjee and Mukherjee (1953) studied the iron metabolism in pregnancy. They found that in normal pregnancy total iron binding capacity gradually increases from the third month onwards till at term it has increased by 59·5 μg./100 c.c. serum. Saturation percentage falls 14·7 per cent. In anaemias of pregnancy they found an increase in total iron binding capacity in microcytic hypochromic, dimorphic and macrocytic hyperchromic anaemias. Agarwal and Misra (1955) estimated serum iron and iron binding capacity in iron-deficiency anaemias, anaemias of infection, liver disease, kidney disease, leukaemias and malignant neoplasms. They found an increase in serum iron binding capacity in iron-deficiency anaemias and leukaemias. Its value was, however, lower in states of infection, cirrhosis of liver, kidney disease and malignant neoplasms.

Intravenous Iron Tolerance Tests

Intravenous iron tolerance tests have been recently described. They permit the measurement of fasting serum iron level, as the first specimen, total iron binding capacity as the second specimen taken 5 minutes after the intravenous injection of ferric ammonium citrate or any other easily ionisable inorganic iron salt, and 120 minute serum iron level as the third specimen. The ratio serum iron/total iron binding capacity, represents saturation percentage, the increase from the fasting serum iron level to total iron binding capacity is the unsaturated iron binding capacity, and the decrease in the serum iron level from the 5 minute specimen to 120 minute specimen is apparently the function of tissue uptake of serum iron. In normal subjects there is a fairly constant decrease/increase ratio. The results of these tests are affected by liver disease, malignant neoplasms, haemosiderosis, severely altered protein metabolism and various haematological disorders. Each test should therefore be evaluated in the light of these variable factors, and interpreted in their entirety taking into account the clinical and laboratory data of each individual patient. Gitlow and Bayers (1952a) state that the test is of value in differentiating the cirrhosis of liver from haemochromatosis. Totterman (1951) obtained flat type of curves in malignant neoplasms.

Plasma and Red Cell Iron Turnover

Huff and others (1950) studied the plasma and red cell iron turnover in normal subjects and patients with various haemopoietic disorders. They state that plasma iron turnover is increased in polycythaemia, leukaemia, pernicious anaemia and haemolytic anaemias. Likewise, in myeloid leukaemia the red cell iron turnover rate was increased in some instances.

Storage and Distribution

It is not yet fully established as to how the iron leaves the blood stream. There are two views about it: (1) That iron leaves the blood stream as iron-transferrin complex (2) That iron leaves the blood stream in an ionised form. Laurell (1951) states that there is more evidence to suggest that iron leaves the blood stream in an ionised form.

The iron carried over by the plasma is distributed all over the body. 60-70 per cent of this iron is found in combination with porphyrin in the form of haeme compounds. These include blood haemoglobin, myoglobin and various haeme enzymes like catalase, peroxidase and cytochrome. The rest of the iron is in available iron reserves like liver, spleen and bonemarrow. This is stored chiefly in the form of ferritin or haemosiderin. Hynes (1949) showed that the reserve amounted to 600 mg. iron. The iron taken up by liver, spleen and bonemarrow is stored mainly in the form of ferritin. Hahn (1948) considered the possibility that ferritin may be identical with haemosiderin. It is generally thought that haemosiderin is an iron globulin complex like ferritin. But the protein moiety of ferritin that is apoferritin has a molecular weight of 460,000 whereas the protein moiety of haemosiderin has a molecular weight of 68,000 only. It is quite likely that the former may be formed by the aggregation of the latter.

Clinical Evaluation of Iron Stores

Stevens, Coleman and Finch (1953) stated that marrow examination for haemosiderin is a reliable index of iron stores. It is frequently the decisive test in the diagnosis of iron-deficiency anaemias. Iron content in Prussian blue stained slides is graded as none, slight, moderate or heavy. In normal subjects iron is slight, in iron-deficiency anaemias slight or none. In pernicious anaemia, cirrhosis of liver, anaemia of infection, and haemolytic anaemias iron is increased in stores. Hutchinson (1953) also estimated the amount of stainable iron in histological sections of bonemarrow. Marrow devoid of iron was found in iron deficiency anaemias and chronic haemorrhagic anaemias, while in aplastic and haemolytic anaemias it contained an excess of iron.

Utilisation

Greenberg *et al* (1947a) studied the fate of injected radioactive iron in presence of inflammation. They concluded that the utilisation of iron for haemoglobin synthesis is impaired in presence of inflammation. Finch *et al* (1949) studied the pathophysiology of iron storage. They injected radioactive iron Fe^{59} and Fe^{59} into normal and pathologic subjects. Their observations showed diminished utilisation of iron for haemoglobin synthesis in refractory, aplastic, myelophthisic anaemias as well as in infection.

Excretion

Iron excretion is small and insignificant. Practically most of the iron is excreted in bile and urine. Iron output in faeces is largely determined by the iron content of the food which is not absorbed. Urinary excretion amounts to 0·5 mg. daily for women and 0·4 mg. daily for men. Biliary iron excretion has been studied by means of biliary fistulae. It has been concluded that 3 per cent of the iron derived from the effete corpuscles is excreted in bile (Hawkins and Hahn, 1944). It is seen that under conditions of acute intravascular haemolysis biliary excretion of iron is increased in proportion to the extent of haemolytic process. In contrast, the iron which is injected intravenously does not increase biliary excretion. It is thus logical to conclude that biliary iron excretion is entirely associated with pigment excretion. That the iron excretion in the faeces is very small was demonstrated by McCance and Widdowson (1937) by observing the effect of rapid haemolysis of red cells on iron excretion in a patient of polycythaemia who was treated with phenyl hydrazine. In other experiments observing the fate of haemoglobin liberated from the rapid haemolysis of transfused blood, the same authors showed that the faecal output was almost identical with the dietary intake and contained no supplement from the haemolysed cells. In women menstrual loss may result in an iron loss of 10-40 mg. each month. Mitchell and Hamilton (1949) found that sweat contains 1-2 mg. iron per litre and constitutes as serious an iron loss as menstrual bleeding.

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CASE NOTE

CONSTRICTION RING DYSTOCIA IN THE LOWER UTERINE SEGMENT

J. C. CHATTERJEE, M.O.,

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Constriction ring dystocia is a recognised clinical entity. Difficulty in its diagnosis lies in the fact, that the site of the constriction has often to be presumed and cannot be confirmed until one can introduce the fingers or if it is higher up, the whole hand through the cervix. The last measure is possible only where the cervix is fully or nearly fully dilated.

This explains the variation in the incidence as quoted by various authors. Usually the constriction ring appears at the junction of the upper and lower uterine segments, but it may appear only in the upper segment, or in the lower segment or in the cervix. The constriction occupying the whole of the lower uterine segment seems to be a rarity, and its occurrence has been doubted by many authors.

It is believed that localised constrictions may occur in any part of the uterus, but it is extremely doubtful if an actual stricture can form in the lower segment. Recent studies of Borell and Fernström (1952) confirm the existence of a functional sphincter at the isthmus uteri. These authors have even observed that this sphincter is influenced by oestrin and progesterone. Although no histologically significant difference can be observed between the corpus and isthmus uteri, a difference in the biochemical structure has been observed by Naeslund and Snellmann (1951). While eliciting electric potential signals during labour it was found by Halliday and Heynes (1952) that the signal from lower uterine segment often equalled to and in a few cases exceeded that from the upper segment. Constriction ring dystocia possibly does not develop all at once and probably represents the ultimate terminal phenomenon of a clinical condition known as hypertonic lower uterine segment. This clinical variety of incoordinate uterine action is fairly common. The upper uterine segment is the site of feeble action while the lower uterine segment is the site of hypertonus as if polarity of the uterine action has been reversed.

The condition can be diagnosed by palpation when placing two hands simultaneously on the body and fundus, and the lower uterine segment. Distention of the intestines specially of the colon is often a marked feature and the uterus may be seen to have been pushed to one side exaggerating the dextrodeviation of the uterus. The cervix remains thick and dilatation is retarded. The hydrostatic pressure gliding down the conical lower uterine segment cause an early rupture of membranes and although there remains some space between the cervix and the presenting part caput formation is the rule. The patient is in agony due to infrequent contractions though a persistent distress is a rule rather than an exception.

In the Fraser Hospital from September 1953 to September 1954 there were altogether 2061 confinements, and hyperactivity of the lower uterine segment was present in 7·5 per cent of cases.

Absolute constriction in the whole of the lower uterine segment was observed during a caesarean section done on 23-6-52 in the Nil Ratan Sircar Medical College Hospital, Calcutta. The interesting observations in this case are as follows :

1. Constriction occupying the whole of the lower uterine segment gripping the neck of the baby which therefore appeared elongated.

2. There was high amputation of the cervix done on this patient about 2 years previously which produced an almost complete occlusion except for a small opening which could be palpated just under the medial aspect

of the left inferior ramus of the pubis in the lateral fornix of cervicovaginal vault.

3. Area below the constriction usually remains flabby and hangs like a curtain, but as the whole of the cervix appeared like a shallow trough on the vaginal vault it was not apparently flabby; on the other hand, the presenting head pushed the trough down and stretched it.

4. Just above the area of constriction in the lower segment there were thick muscular fibres of the upper uterine segment, thus proving that the area of constriction ring dystocia was in the lower uterine segment.

Case Report

Mrs. S. B. D., aged 26, was admitted in the Nil Ratan Sircar Hospital on 23-6-1952 at 4-30 A.M. She was a fifth gravida with previous obstetric history normal. The patient had a high amputation of the cervix done about a year back.

On examination she appeared to be a thin individual carrying full term. History showed that the labour was overdue by 11 days. The pain started in the early hours of the previous morning.

Anaemia was not marked. The pulse rate was 84 per minute.

Abdominal examination revealed a full term uterus. The fundus and the body were expanded while the lower part enclosing the neck and the head was narrow. The head was not deeply engaged but fixed. Foetal heart sounds were present but the rate was 164 per minute. Examination per vaginum revealed that the vault of the vagina was occupied by a shallow trough where no cervical opening could be detected. A small opening could however be made out on careful examination behind the symphysis at about the 2 o'clock position. It was a pin point opening not admitting even the tip of the index finger.

This shallow vault of the vagina was occupied by the head which was trying to engage into the pelvis. There was no apparent cephalopelvic disproportion and the head could be made to engage when pushed from above. A caesarean section was decided upon. On laparotomy the neck of the baby appeared elongated as the whole length of the lower uterine segment was gripping around the neck, while the body and fundus looked like an expanded flask with narrow neck represented by the lower uterine segment.

A transverse incision was made into the middle of the lower uterine segment over the constricted area. But head could not be delivered through the rent and a longitudinal incision like the vertical limb of a T had to be made extending into the body of the uterus. The baby was extracted out of the uterus. There was very scanty liquor amnii which was saffron coloured with sandy deposits over the surface of the membranes as well as the baby. The baby appeared rather listless though there was pulsation in the cord and was revived with some difficulty.

0·25 mg. ergometrine was injected into the uterus and the placenta was separated from the wall of the uterus with membranes. These were also yellowish in colour with gritty sandy deposits in the foetal surface of the placenta.

The cervical canal was shut off from the vagina by a shallow basin like the upper surface of a watch glass and there was almost no communication. A long artery forceps was pushed through the partition and as the blades were separated an opening was made ¼" in diameter between the uterine and vaginal canal to help drainage of lochia.

The uterine incision was closed by double layers of sutures, and the abdomen closed. The puerperium was uneventful.

Acknowledgment

I am grateful to the Superintendent and Principal, Nil Ratan Sircar Medical College and Hospitals, Calcutta for permission to use the case records.

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XXXII ALL-INDIA MEDICAL CONFERENCE, JAIPUR

The 32nd All-India Medical Conference will be held at Jaipur on 26-28 December, 1955. Dr. A. C. Ukil of Calcutta with preside. The meeting of the Working Committee of the Indian Medical Association will be held on 22-23 December, 1955 and of the Central Council, on 24-25 December, 1955

Intending delegates and visitors, attending the Conference either alone or with families are requested to write to the Organising Secretary (with copy to the respective Branch Secretaries) immediately for reservation of lodging and the type of food they would like to take. Railway concession forms will be issued from the office of the Organising Secretary. Those who intend to read paper or take part in the symposium should inform the Secretary, Scientific Transaction Committee by the end of November, 1955 at the latest.

JOURNAL OF THE
INDIAN MEDICAL ASSOCIATION

CALCUTTA NOVEMBER 16, 1955

THE EMPHYSEMA HEART

Rapid advancement in the field of newer methods of investigation of cardiovascular dynamics in the normal and diseased heart has brought a comparatively less known heart disease in the forefront. The symptomatology and treatment of this heart disease are somewhat different from the familiar types with which the general practitioners are acquainted. There is right-sided congestive cardiac failure (decompensation) as a result of gradual narrowing of the vascular bed of the lesser circulation due to chronic disease of the lungs. Thus emphysema heart or chronic cor pulmonale signifies right heart enlargement, strain and decompensation secondary to certain forms of chronic disease affecting primarily the lung parenchyma. The primary difference between congestive decompensation of chronic cor pulmonale and other types is the presence of low arterial oxygen content in the former.

Though less known the condition is the third most frequent heart disease, with an incidence of 16 per cent in Halmagyi's[1] series and 20 per cent in Paul Wood's[2] series.

The commonest cause of chronic cor pulmonale is some morbid change in the lung parenchyma and 80 per cent of the cases is due to chronic bronchitis, bronchial asthma and chronic pulmonary fibrosis all leading to secondary emphysema lungs. How this pulmonary hypertension comes about in emphysema is complex. That it is not caused by a single factor is certain. Several variable factors may be working.

Emphysema of the lung is a progressive disease and with the progress the total respiratory surface is gradually diminished with a reduction of the vital capacity. The alveolar walls become thickened and many capillaries get occluded. These morbid changes negate the primary purpose of the lesser circulation, that is oxygenation of blood. The ineffective alveolar ventilation causing disturbed gas exchange leads to hypoxia with retention of carbon dioxide. The oxygen saturation of the arterial blood may be 75 per cent or less with a rise of interalveolar CO_2 tension to 50-60 mm. of Hg. The respiratory centre becomes less sensitive to raised arterial CO_2 content, and thus the dyspnoea and cyanosis are not proportionate.[3]

That emphysema is the cause of pulmonary vascular sclerosis is disputed. William Evans[4] suggested that rise in pulmonary pressure due to emphysema might lead to hypoplastic changes in the vascular media and endarteritis and eventually a serious pulmonary hypertension. But Welch and Kinney[5] have shown that normally from the fourth decade on, there was some degree of arteriosclerosis in every case examined by them. The incidence of vascular changes as observed by Florence McKeown[6] definitely rules out emphysema as the morphological basis for the pulmonary hypertension. Emphysema might accentuate the normal age changes. The studies on circulatory changes in severe pulmonary emphysema made by Mounsey *et al*[7] and supported by Cournaud[8] suggest a relationship between hypoxia and pulmonary hypertension.

The vessels of the pulmonary circulation are sensitive to hypoxia. The hypoxia at first gives rise to local vascular spasm and as the hypoxia with CO_2 retention increases a diffuse vasoconstriction occurs. Thus a high pulmonary pressure develops, but why then the correction of hypoxia does not cause a reduction of high P.A. systolic pressure? Though not much emphasis is laid on it, another factor probably plays an important role. It is the role of the autonomic nervous system in the production of spasm. Fowler *et al*[9] reported that methonium compounds reduce the pulmonary pressure considerably and Whiteside and Coigby[10] obtained beneficial effects of hexamethonium bromide in emphysema. On the other hand five of Simpson's[11] cases derived no benefit inspite of the effect of this drug on the systemic pressure.

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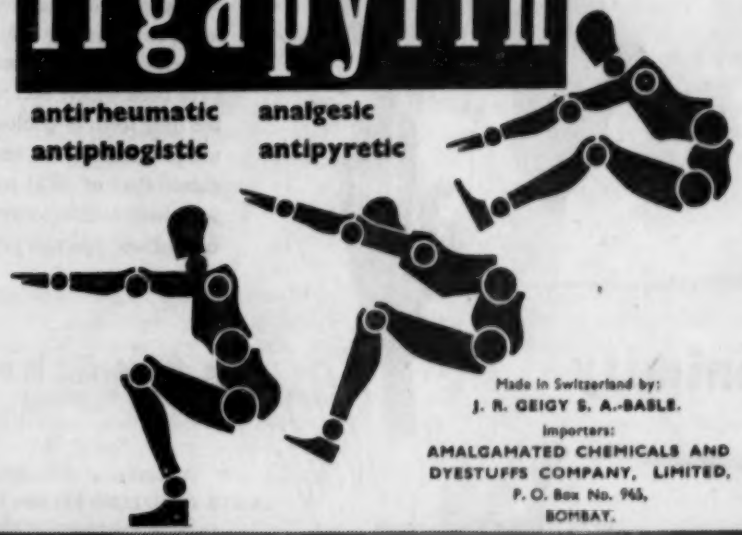

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The impediment of right ventricular function and its strain are a gradual process. In the first stage of emphysema the heart and the circulation are within normal limits. As the disease progresses further hypoxia occurs leading to reflex pulmonary hypertension particularly on exercise (Selzer[12]). In the last stage circulatory failure becomes manifest. This may be gradual, but more often it is precipitated due to an acute bronchopulmonary infection or due to an attack of severe bronchospasm. In these episodes of emphysema heart failure the pulmonary hypertension and enlargement of the right ventricle are transient and reversible (Mounsey *et al*[8] and Simpson[13]).

Skiagram for the diagnosis of cor pulmonale in upright position in the P.A. view is not reliable. The prominence of the pulmonary arteries, an allegedly characteristic sign could be detected only in 30 per cent of Halmagyi's series. In chronic cor pulmonale a small heart with big conus is particularly noticeable. The small heart is apparent. Cochran Shanks and Kerby[14] suggest that a skiagram in supine position will reveal the real increase in the size of the heart.

The cardiographic criteria of right ventricular hypertrophy and strain in chronic emphysema according to William Evans[15] are: a deep S in LI, inverted T_3, depressed S-T in LII, a tall R in right precordial lead and a deep S in terminal left precardial lead. Mounsey *et al*[16] made cardiographic studies on 16 patients with severe pulmonary emphysema. Their criteria were: A prolonged time interval between the beginning of the QRS complex and the onset of the intrinsicoid deflection in V_1, a total duration of QRS complex less than 0·12 sec., a tendency to a small Q wave in V_1, a reversal of the ratio of the amplitude of R and S waves in V_1 and V_6, (large R in V_1 and a prominent S in V_6), a tendency of inversion of T in V_1 to an upright T in V_6 and absence of notching of R in V_1 and with those criteria they found E.C.G. evidences of right ventricular hypertrophy only in 2 cases. In one of their cases gross right ventricular hypertrophy was found at autopsy without any cardiographic evidence of right ventricular hypertrophy. Maurice Campbell[17] in an editorial has stated that a large S_1 and R_3 with insignificant R_1 and S_3 are quite unusual in chronic pulmonary disease. The actual size of R and S waves in V_1 in normal subjects is about 5 mm. and 10 mm. respectively. In pulmonary disease inspite of associated gross right ventricular hypertrophy the R is 4·4 mm. and S 13·3 mm. (Thomas[18]).

Evidences of right ventricular hypertrophy, if they develop, are late events in emphysema heart disease. The value of E.C.G. is more confirmatory than diagnostic. Moreover the pulmonary heart disease with manifest cardiac insufficiency occurs in majority of cases in advanced age, an age where we expect coronary changes. Amongst 11 of the patients suffering from chronic cor pulmonale with failure it was not possible for Lewis *et al*[19] to rule out additional diagnosis of arteriosclerosis in the older age group. The negative T and a depressed S-T may therefore be explained by the fact that chronic cor pulmonale is the disease of age where a certain amount of coronary arteriosclerosis may be present.

The treatment of chronic cor pulmonale is the treatment of the respiratory infection and bronchospasm and right-sided cardiac decompensation. Caution is needed in the free use of oxygen inhalation. To avoid serious respiratory acidosis oxygen inhalation should be intermittent and concentration increased gradually. Digitalis is indicated whenever there is right sided congestive failure, and inspite of the theories against use of digitalis, clinical improvement is noticeable. Immediate effect of digitalis as studied by Mounsey *et al*[7] by cardiac catheterisation in pulmonary emphysema was rise of right ventricular pulse pressure without significant changes in the cardiac output. Recently the use of carbonic anhydrase inhibitor has been found effective in right-sided decompensation due to chronic cor pulmonale.

Pulmonary hypertension in emphysema and consequent right ventricular decompensation are not due to one factor only. The resultant physiological abnormalities are of far greater importance than the structural changes.

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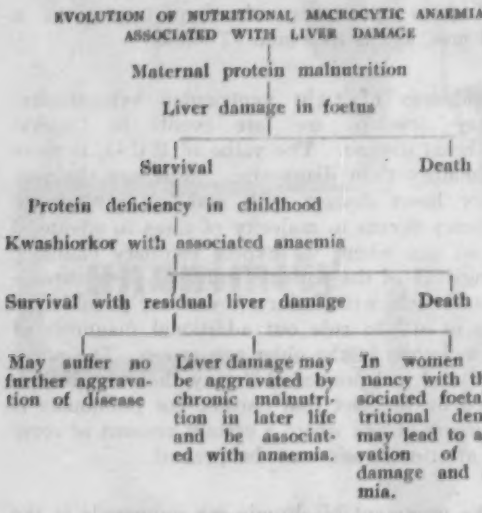
CURRENT MEDICAL LITERATURE

Natural History of Anaemia Associated with Protein Malnutrition

WOODRUFF (*Brit. M. J.*, **1**: 1298, 1955) gives in the following lines the summary of his Goultonian lecture on the natural history of anaemia associated with protein malnutrition:


EVOLUTION OF NUTRITIONAL MACROCYTIC ANAEMIA ASSOCIATED WITH LIVER DAMAGE
Maternal protein malnutrition
Liver damage in foetus
Survival
Death
Protein deficiency in childhood
Kwashiorkor with associated anaemia
Survival with residual liver damage
Death
May suffer no further aggravation of disease
Liver damage may be aggravated by chronic malnutrition in later life and be associated with anaemia.
In women pregnancy with the associated foetal nutritional demands may lead to aggravation of liver damage and anaemia.

Among certain communities children may be born with livers already damaged by maternal malnutrition. If they survive they again pass through a period following weaning during which the demand for protein imposed by growth is greater than the supply, and the liver is liable to be damaged. At these times an anaemia and other signs of malnutrition may develop. The anaemia is associated with a macronormoblastic marrow and with red cells which are broader and thinner than normal. Many succumb while passing through this stage in childhood; others recover, but probably the majority of these latter are left with some hepatic damage and fibrosis. Nevertheless the functional reserve of the liver is such that, in spite of this, the patient manages until he or she passes through a period in which demands for protein are again in excess of the available supply. In women this state may occur in acute form during pregnancy, foetal growth being the precipitating factor. In adult males and non-pregnant females the disease is less common, but when seen in hospital practice in Nigeria it usually represents a late stage of a chronic process. It develops if the diet is markedly and chronically deficient in protein, and particularly if parasites impose an additional drain on deficient supplies of nutrients. In these cases one of the factors in the causation of the anaemia may be that haemoglobin in its production can draw on plasma proteins, as has been shown by Whipple, and thus when these proteins are abnormal or deficient the production of haemoglobin may be affected. In these cases abnormality, as distinct from deficiency, of plasma proteins may arise from the production of antibodies containing protein, or from liver disease, it being established that the liver plays an important part in protein synthesis.

The characteristics of protein malnutrition in Nigerians are similar whether it occurs in childhood, in pregnancy, or in adult life unassociated with pregnancy. In many cases the disease starts in early life, or even in intrauterine life, and progresses as it is aggravated during periods of growth or deprivation until finally it ends in advanced hepatic cirrhosis with all its concomitant haematological and other features. This stage may even be reached in childhood.

Many of the clinical and pathological states considered in this work are gross and unlikely to be encountered in Great Britain. Nevertheless they provide an insight into the function of various organs and tissues, and into processes which may affect the body in a less florid form in the temperate zones in conditions—such as nephrosis, exudative dermatoses, burns, alcoholism, and cirrhosis of the liver—resulting from non-nutritional causes.

A B O Blood-Groups and Gastric Acidity

KOSTER AND OTHERS (*Lancet*, **2**: 52, 1955) write:

The A B O blood-groups have been investigated in 680 cases of duodenal ulcer, 337 cases of gastric ulcer, 105 cases of gastric carcinoma associated with free hydrochloric acid in the stomach, and 195 cases of gastric carcinoma associated with achlorhydria. The controls were 14,304 healthy people from the same locality.

The patients with duodenal ulcer, in whom the secretion of hydrochloric acid was greatest, included 50·2 per cent with group O and 38·4 per cent with group A.

In patients with gastric carcinoma associated with achlorhydria 33·2 per cent were of group O, and 56·4 per cent were of group A.

The frequency of blood-groups B and AB was not similarly reversed.

Ventricular Aneurism of the Heart

VAKIL (*Am. Heart J.*, **49**: 934, 1955) from an analysis of the observations on twenty proved cases of ventricular aneurysm describes the following clinical signs, hitherto undescribed in the literature, namely, (1) an anomalous location of the cardiac impulse (50 per cent of cases); (2) a myotonic cardiac impulse (65 per cent), (3) a double thrust or reduplicated impulse (20 per cent), (4) a wavy impulse or cardiac shudder (30 per cent), (5) a heaving left costal margin, (6) stony dullness over the precordium, and (7) a loud and long, musical or "cooing" systolic-diastolic murmur. These signs are briefly described with a view to stimulate further inquiry into the matter.

Depression by Quinidine

FINNEGAN AND TROUNCE (*Brit. Heart J.*, **16**: 341, 1954) observe that amongst 115 patients treated with quinidine for cardiac arrhythmia at Guy's Hospital, London, 3 developed severe reactions, which were fatal in one. The main changes noted electrocardiographically in these cases indicated depression of the bundle and nodal tissue. In 5 further cases electrocardiographic

abnormalities were noted in the absence of symptoms of collapse. These abnormalities again indicated depression of various parts of the nodal or conducting tissue, but in some cases ventricular extrasystoles or tachycardia were present in addition. It was found that the experimental administration of quinidine to rabbits produced effects similar to those seen in man. Various substances—adrenaline, noradrenaline, phenylephrine, caffeine ephedrine, and acetylcholine,—were used in an attempt to revive the heart depressed by quinidine, adrenaline being the most effective both in the isolated perfused heart and in the intact animal. The application of these findings to the treatment of cardiac arrest in man following the administration of quinidine is discussed.

Lung Lesions in Rheumatoid Arthritis

ARONOFF AND OTHERS (*Brit. M. J.*, **2**: 228, 1955) give in the following lines the summary of their observations:

The case histories of 253 patients with rheumatoid arthritis, chest x-ray findings of 130 of these and of 130 non-rheumatoids, and post-mortem findings of 42 rheumatoid cases and 42 controls were reviewed to determine the incidence of pulmonary lesions in a rheumatoid and non-rheumatoid series of patients.

1. No specific lung lesion was found which could characteristically be associated with rheumatoid arthritis.

2. The clinical and post-mortem incidence of various non-specific pulmonary lesions appeared to be rather greater in the rheumatoid patients than in the control series, but no one particular lesion was outstanding.

3. In patients with clinical and radiological evidence of pulmonary disease, no temporal correlation was observed with exacerbations of rheumatoid disease.

4. In this comparatively small group of patients studied, no example of what has been described as "rheumatoid lung" or "rheumatoid lung syndrome" was observed.

5. Four cases are cited in detail, illustrating what have been in our experience the most difficult of the problems of differential diagnosis of lung lesions in rheumatoid arthritis.

Aspirin and Ulcer

MUIR AND COSSAR (*Brit. M. J.*, **2**: 7, 1955) write that aspirin is a serious gastric irritant, particularly in peptic ulcer patients.

It plays a significant part in exacerbation of ulcer symptoms and is at least a contributory cause in one in eight cases of haematemesis and in one in five of that group with no major dyspeptic history.

Effect of Oxytetracycline in Streptococcal Sore Throat on Incidence of Acute Rheumatic Fever

CATANZARO AND OTHERS (*Ann. Int. Med.*, **42**: 345, 1955) from an analysis of the results of treatment of 506 patients with streptococcal exudative pharyngitis with oxytetracycline administered for five days (four hundred eighty patients who received no specific therapy serving as control) observe:

In the dosages employed, oxytetracycline reduced the convalescent streptococcal carrier rate 55 per cent as compared to the control group. In addition, the average antistreptolysin response in the treated patients was 49 per cent less than that observed in the control patients. Maximal inhibition of antibody production occurred in the treated patients in whom the infecting organism was eliminated.

Recurrent pharyngitis occurred more frequently in the treated group, particularly in those patients who remained carriers. The second clinical illness usually developed within two to three weeks after therapy had been discontinued. Suppurative complications were of infrequent occurrence in both groups.

Rheumatic fever developed within 35 days after the onset of the streptococcal infection in 12 patients in the control group and in five patients in the group treated with oxytetracycline. The attack rate was reduced, therefore, from 2·94 per cent in the control group to 0·99 per cent by therapy with oxytetracycline.

Dientamoeba Fragilis, an Intestinal Pathogen

SWERDLOW AND LURROWS (*J.A.M.A.*, **158**: 176, 1955) write that the frequency with which *Dientamoeba fragilis* is found in the appendix, its preference for red blood cells when they are available, and the concomitant occurrence of fibrosis of the appendiceal wall in all cases of *D. fragilis* infections of the appendix incriminate it as a pathogen. Nearly half of the observed cases were in children, an age group that rarely shows a fibrotic condition of the appendix. Inasmuch as this amoeba causes various clinical symptoms, ingests blood cells, and causes tissue changes in the host, infections should be treated whenever encountered.

Acetazoleamide in Acute Respiratory Acidosis

WISHART AND ISAACS (*Lancet*, **1**: 995, 1955) write that the carbonic anhydrase inhibitor, acetazoleamide, was given to 3 patients with acute exacerbation of chronic respiratory acidosis.

Increased urinary excretion of bicarbonate and a fall of the serum-bicarbonate level resulted.

In all 3 cases there was clinical deterioration. 2 of the patients died, and the 3rd recovered when the acetazoleamide was withheld.

It is concluded that acetazoleamide may be detrimental in acute respiratory acidosis.

Diaphragmatic Breathing in Chronic Pulmonary Emphysema

MILLER (*Am. J. Med.*, **17**: 471, 1954) from the study of the effect of diaphragmatic breathing in 24 patients with chronic pulmonary emphysema observes:

The results showed that diaphragmatic breathing produced a striking increase in tidal volume at a lower respiratory rate, a lowering of the respiratory midposition and more effective alveolar ventilation, with increased arterial oxygen saturation and decreased carbon dioxide concentration. There was no significant increase in total ventilation, except when this had been initially diminished. The author concludes that training

in diaphragmatic breathing produced improvement in pulmonary function, and that this is associated in many cases with a more effective cough mechanism, resulting in better evacuation of bronchial secretions.

Respiratory Acidosis in Emphysema

COHN AND OTHERS (*Am. J. Med.*, **17** : 447, 1954) after a study of 8 emphysematous patients, 5 with acute respiratory acidosis and 3 with chronic acidosis observe :

In the acute cases pulmonary infection and acute heart failure were superimposed, and in the chronic cases cor pulmonale was present.

Treatment which included administration of bronchodilator drugs, antibiotics, and oxygen and breathing exercises or the use of mechanical respirators, was successful in reversing the acid-base abnormality in the acute cases but not in the patients with chronic carbon dioxide retention. It is pointed out that the main dilemma in the treatment of these cases consists in the fact that the patient suffers from both hypoxia and hypercapnia, and that treating the former with conventional oxygen therapy increases the severity of the latter.

Dysplenism

REINHARD AND LOEB (*J.A.M.A.*, **158** : 629, 1955) in dealing with dysplenism secondary to chronic leukaemia or malignant lymphoma observe :

The fact that a given patient with chronic lymphocytic leukaemia or malignant lymphoma has a disease that is known to be capable of damaging the bone marrow and eventually resulting in anaemia, thrombocytopenia, or leukopenia should not blind the clinician to the possibility that depression of one or more of these cellular elements in the blood may be mediated through a splenic mechanism. If it can be demonstrated that the patient is destroying red blood cells at an abnormally rapid rate, or if the patient has a thrombocytopenia or granulocytopenia in spite of a marrow that appears to be capable of producing those cells in adequate numbers, splenectomy should be seriously considered. At times, this therapeutic approach can be of significant value from a palliative standpoint even though the patient has far-advanced malignant disease.

Roentgen Examination of the Acute Abdomen

YOUNG (*Radiology*, **64** : 483, 1955) in the Carman lecture mentions that roentgen examination of the abdomen reveals manifestations of many acute abnormalities and therefore it is an invaluable aid in their recognition and evaluation. It is of great value in demonstrating the site and frequently the character of the acute process, thus enabling prompt and effective treatment. This information is often made available by plain-film examination, but a contrast medium is occasionally necessary.

The roentgen findings in many acute abdominal conditions are discussed, including mechanical, vascular and paralytic ileus; volvulus; intussusception; external and internal hernia; perforation of a hollow viscus, with resultant gas in the peritoneal or retroperitoneal spaces; calculous disease, and inflammatory, cystic, and neoplastic enlargements of the gallbladder, pancreas, and kidney; appendiceal, pelvic, and subdiaphragmatic abscess; rupture of the kidney and spleen.

CURRENT TOPIC

MEDICAL SERVICES IN THE REPUBLIC OF IRELAND†

P. J. DELANEY,

Secretary, Irish Medical Association.

INTRODUCTION

In the Republic of Ireland, as in most other countries, there has been a growing intrusion by the state into almost all fields of medical practice with consequent encroachment of state influence and control over these services which had formerly been provided by voluntary agencies.

While medical practice in Ireland has not become as "socialized" as in a number of other European countries the threat of more state interference, actuated as it is by political expediency, remains a constant danger.

The Irish Medical Association supported by the more educated and thoughtful classes has, with varying success, vigorously opposed this state paternalism. The association has always expressed its desire for the best possible services conditional on full clinical freedom and other necessary safeguards.

HOSPITAL SWEEPSTAKES

No survey of the medical services in Ireland would be complete without some information on the Irish Hospital Sweepstakes Fund. Since the Hospital Sweepstakes were instituted in 1932, 30½ million pounds have been given to the hospitals of which roughly 18 million pounds have been given to voluntary hospitals to help them meet their deficits, and 13 million pounds have gone to state hospitals. In the beginning the sweepstakes were instituted to aid the voluntary hospitals only, but after a few years when it was found to be so successful the state entered into the situation and started aiding the state hospitals from the fund. In addition the state has applied a 30 per cent stamp duty on the tickets and has taken in 7 or 8 million pounds by this method. There are about 10 million pounds in the sweepstakes treasury. Prior to the sweepstakes, the private hospitals were financed by grants and gifts from private individuals but these sources have gradually ceased, due to the economic situation of the individual. Costs for maintenance and drugs continue to increase. Hospitals have a deficit each year and are paid by the sweepstakes. The government has complete control and charge over the administration of a private hospital.

There is a difference of opinion on the sweepstakes. Some regard it with disfavour as a national lottery for the solution of an urgent problem. This is true and the government uses this fact as a means of pressure upon the medical profession. It is unfortunate and creates a social problem. A summary of the legislation which curtails the doctor proves the point.

Present medical services may best be appreciated by dividing the population (approximately 3,000,000) into

† *World Medical Journal*, **2** : 201, 1955.

three classes—the "necessitous poor", the middle income group, and the upper income group. The total population is almost equally divided into these three groups.

The Necessitous Poor

In Ireland there is no definition or income level set to indicate a "poor person." Any person who by his own effort is unable to provide medical service for himself or his family can be classified as "necessitous poor." The tickets are issued by the local wardens. A large majority of the patients never have a ticket. The doctors know the patients, their circumstances and the conditions and, therefore, they are treated free of charge.

For this class a full range of domiciliary, consultative and hospital services is available free of all charge. Owing to difficulty of interpretation of the term "necessitous poor" considerable abuses exist under which large numbers, not strictly entitled to this "free" service, avail themselves of its provisions.

Each area has a part-time dispensary doctor who is on call to aid all those people classified as the "necessitous poor." It is his duty to attend them day or night without charge. If their need is beyond his capacity he can recommend any form of treatment he desires for them. Domiciliary care is provided by part-time salaried District Medical Officers plus nursing auxiliaries. Consultant and hospital care is provided by District or Cottage Hospitals, large County Hospitals and, where specialised attention is required, by the voluntary teaching hospitals. For this group all medications are supplied free.

Thanks in large measure to funds acquired from the Irish Hospital Sweepstake Funds a great extension of state supported hospitals has taken place staffed by physicians and surgeons who are largely whole-time and in this way local consultative services at a high level are available throughout the country.

Here it should be stated that all medical appointments to the local or central authority services are made through a statutory body known as the Local Appointments Commission on whose selection boards the profession has very liberal representation.

Ireland has, in addition to its medical service, an extensive social service which has its own funds which are administered by the local authorities. This provides actual needs at a low level.

Middle Income Group

Under recent legislation the middle income group will be entitled to "free" or almost "free" consultant and hospital services. Except in the case of maternity services they will still have to pay for domiciliary treatment, as it is felt by all concerned that in this respect there will be no undue hardship.

Upper Income Group

No "free" treatment is envisaged for people in the upper income group except that their children up to six years of age will be entitled to "free" treatment at Child Welfare Clinics, families will be entitled to "free" maternity services on payment of £1 per year, and all will be entitled to free institutional treatment for scheduled infectious diseases.

In addition certain people in this class may, by virtue of special circumstances, apply for provision of free consultant and hospital services, provided the local authority is satisfied that their claims are genuine.

Number of Doctors

Approximately 2,500 doctors are in active practice in Ireland and this number is considered adequate having regard to the fact that the country in general is so lightly industrialised.

Of the doctors, 1,500-1,600, are family doctors or general practitioners, about 650 of whom are district medical officers paid by the local authorities and are part-time. The remainder are private practitioners. About 500 consultants are whole-time medical officers of health, mental hospitals, etc. The whole-time officer is provided to give medical service to the indigent class but in most cases they are allowed to have a small number of private beds for their private patients. However, they may not use these beds if they are needed for the indigent class. Specialists are provided for the indigent in the hospitals free of charge. This may be altered with the new legislation.

Remuneration

The average full-time hospital doctor, a specialist with 35 years of practice, receives a salary of from 1,200 to 2,000 pounds a year and may also have a free house. This varies with the size of the hospital.

The rate of fees charged by the private practitioner varies from district to district and from family to family. The average would probably be between 5 and 10 shillings for a home visit by the family doctor, the rate being lower if it were near and more if it were far for the doctor to travel. Specialists receive a somewhat higher fee, although this depends a good deal upon the doctor who refers them. The rate could be from 10 to 50 pounds depending on the circumstances. Ireland is a small country and the rate is frequently adjusted between the patient and the specialist. The average income of a general practitioner is from 1,400 to 1,500 pounds a year depending upon whether he is in the city or in the country.

Consultants do not hold positions on a hospital staff. They serve the hospital patients free of charge and charge a consultation fee only when the patient is seen in the office, home, or nursing home.

Medical Education

The medical schools of Ireland educate a larger supply of doctors than the country needs and an increasing number of students are electing to study medicine. At the present time about 80 per cent of the graduates from medical school must be exported to other countries. The medical schools are subsidized from state funds as the fees charged the students are insufficient to cover the expense. Indirectly sweepstakes money is diverted toward medical education as the deficit of teaching hospitals is made up from the sweepstake fund. The number of foreign students in the medical schools is strictly limited. These students come from various parts of Africa, Trinidad, Canada, and the United States and make up less than 5 per cent of the medical school enrollment.

The possibility of limiting admission to medical school or any other branch of learning in Ireland is subject to public controversy. Such a limitation would be an infringement on personal liberty. The standards of education and examinations are being improved. The social wastage through exportation of the educated portion of the population, not only in medicine but in all fields is a serious national problem. Cooperation is being sought to save this national heritage. The number of doctors in practice in Ireland has been increased and the percentage of wastage reduced. However this wastage still amounts to 40 doctors annually.

Hospital Services

In general it is felt that sufficient hospital beds are available in the various Voluntary and State Hospitals for every day requirements except for those needed for mental patients. None the less a big programme of hospital construction is under way to replace outmoded institutions and provide specialist facilities for diseases and disabilities requiring specialist care and attention.

Infectious Diseases

As noted above all persons are entitled to "free" institutional treatment if suffering from any of the scheduled infectious diseases, whilst in addition special weekly grants are made to the families of patients in the lower income groups.

Voluntary Insurance

Some years ago, dissatisfied with the existing medical services then available in this country, and recognising that for many responsible and conscientious members of the community the cost of consultant and hospital services might constitute a major financial and domestic catastrophe, the Irish Medical Association put forward for government consideration a scheme of voluntary state-aided contributory insurance to cover these services. The scheme was not found acceptable by the government then in power. Following a change of government, the present Minister for Health recently appointed an Advisory Committee widely representative of all interests concerned, to consider the feasibility of introduction of such a scheme. On this committee, the Irish Medical Association has direct representation.

Social Services

A wide range of Social Services exists whereby provision is made for pension or allowances to persons in the following categories—all over 70 years of age, blind persons, widows, unemployed, disabled. Payment in respect of some of these is subject to a "means" test. Children's allowances are paid to parents where the number of children is in excess of two.

Conclusion

The medical services of Ireland are a topic of constant debate and vexing controversies. The medical profession is accused or complimented on bringing about the defeat of one government. The medical profession constantly stresses that it favors the best quality of medical service that the country can provide and that good health services cannot be purchased at bargain sale prices. The medical profession of Ireland feels it is making great progress with the current Minister of Health.

NOTES AND NEWS

The World Medical Association

The 9th General Assembly of the World Medical Association was convened in Vienna, Austria, September 20-26, 1955.

Prof. Dr. A. Spinelli of Italy presided at the opening session and inaugurated Dr. Karl Niedergerger of Austria as President for 1955-1956. Following an amendment of the Constitution and By-Laws to provide for a Presiding Officer during the business sessions of the Assembly, Dr. L. A. Hulst of the Netherlands was elected to act during the 9th General Assembly.

Dr. A. Fernandez-Condé, President of the Cuban Medical Association was elected President-Elect for 1955-1956 and the three Council vacancies for 1955-1958 were filled as follows: Dr. L. A. Hulst (Netherlands), Dr. Dag Knutson (Sweden), Sir Lionel Whitby (United Kingdom).

The venue of the 10th General Assembly was set for Havana Cuba to be convened October 9-15, 1956.

University of Vienna Confers Honours

During the 9th General Assembly session, the University of Vienna presented a gold key to Dr. Dwight H. Murray, President-Elect of the American Medical Association and to Dr. Louis H. Bauer, Secretary General of the World Medical Association, in recognition of the contribution these two physicians had made to the progress of medicine.

Burma Medical Association Presents Key

During the visit of Dr. Louis H. Bauer, Secretary General of the World Medical Association to the Burma Medical Association headquarters in Rangoon, he was made an honorary member of the Burma Association receiving from it a gold membership key.

International College of Surgeons

The Tenth International Congress of the International College of Surgeons will be held in Chicago, Illinois, September 9-13, 1956 in conjunction with the twenty-first annual congress of the United States and Canadian sections. The Buenos Aires meeting, previously announced has been cancelled.

Further information relative to this Congress may be obtained by writing to Dr. Max Thorek, International Secretary General, International College of Surgeons, 850 West Irving Park Road, Chicago 13, Illinois.

Nobel Prize for Medicine

The Caroline Medical Institute awarded this year's Nobel Prize for Medicine to Professor Hugo Theorell, a leading Swedish blood specialist.

The 52-year-old professor, head of the Nobel Institute for Biochemistry in Stockholm, is the second Swede to receive the medicine prize and is honoured for 'his discoveries concerning the nature and mode of action of oxidation enzymes'.

He is said to have come close to some of the foremost mysteries of life itself by explaining important features

in the complicated mechanism of cell division and metabolism of cells.

The research for which he was given the prize dates back to 1934 when he succeeded in producing for the first time in pure form the so-called 'yellow enzyme'.

Dr. A. C. Ukil Elected I.M.A. President

At the meeting of the Working Committee of the Indian Medical Association held at Trivandrum on the 14th, 15th and 16th October 1955, it has been announced that Dr. A. C. Ukil of Calcutta has been elected president for the forthcoming year. He will preside over the 32nd session of the All-India Medical Conference to be held in Jaipur on 26th, 27th and 28th December, 1955. Dr. P. R. Trivedi of Ahmedabad, Dr. G. V. Joshi of Hubli (Maharashtra and Karnatak) and Dr. Amiya Kumar Bose of Calcutta have been elected Vice-Presidents of the Association for the year 1955-56. Dr. Ukil and the Vice-Presidents will assume office on the 26th December 1955 at Jaipur.

Big Changes in Medical Education

Important recommendations affecting the system of medical education in India have been made by three sub-committees charged with the task of preparing "working papers" for the Indian Medical Education Conference, opening on November 19.

The complete stoppage of private practice by teachers serving in medical colleges has been strongly recommended as a "desirable goal", by one of the sub-committees. The reason why the staff of medical college go in for private practice is attributed to insufficiently attractive salaries offered by universities and State Governments. Only wholetime staff should be recruited by medical colleges, and the scales of salaries should be generously revised.

The whole pattern of medical education, which is modelled after British universities, has been described in the recommendations as being 'outmoded' by experts who say that even in the U.K. the curricula and methods of teaching have also changed during the last 15 years. To bring the syllabi in medical colleges more in line with modern American and continental practice, a number of changes have been suggested. The most notable of these relates to shifting the present emphasis from written examination to *viva voce* tests.

Another recommendation seeks to reduce the existing period of study for the M.B.B.S. degree to four and a half years. At present the periods vary from five to six years.

This suggestion takes into account the growing need of medical men in the country during the Second Five-Year Plan period and the costly system of education.

A uniform system of admission into medical colleges is also urged. Some colleges take into consideration the past academic records of students before admitting them. Others hold competitive examinations.

The sub-committee on syllabi has pleaded for re-orientating medical education to suit the needs of rural areas. It has been suggested that during the clinical period of training, students should visit rural areas and serve the local population. During the period of internship, every student must spend at least three months in a village dispensary.

Foreign Doctors in India on Tour

Altogether fifty-four doctors and their wives, from twelve countries of Europe and North America, are coming to India on a one-month tour. The object of this tour is to strengthen links between India and the rest of the world.

Malaria Control in Second Plan

The Planning Commission's Health Panel is discussing a scheme, evolved by the Health Ministry and the health experts of the Commission, to extend during the second Five Year Plan protection against malaria to 200 million people who are exposed to the risk.

Likely to cost about Rs. 30 crores, the plan involves the setting up of 38 new malaria control units in addition to the existing 162, established under the first Plan.

It has been estimated that about 100 million suffer from malaria every year. Of these about 71 million die.

When the national malaria control programme was drawn up, it was thought that intensive D.D.T. operations for three years and a maintenance programme on a small scale thereafter would effectively control malaria in the epidemic areas.

The Malaria Institute of India now considers that another five-year programme of active operation is necessary before the changeover to the maintenance stage.

CORRESPONDENCE

The Editor is not responsible for the views expressed by correspondents

B. C. G. Vaccination

SIR,—We have gone through your editorial dated October 1, very carefully and we would like to comment on the procedure adopted in this part of the country, for B.C.G. Vaccination.

Some of the workers on this problem suggest compulsory tuberculin test of B.C.G. vaccinated persons, three months afterwards, in order to determine tuberculin allergy, which indicates presence of immunity in B.C.G. vaccinated persons. If found negative, it is essential to re-vaccinate with B.C.G., till tuberculin allergy to a requisite dilution develops, in order to avoid any false sense of security for immunity against such a dreadful disease. It has been advised "to repeat B.C.G. vaccination if original inoculation fails to render the person tuberculin sensitive."

Last year, a son of one of the writers' of this letter, aged about 8 years, having been found to be a negative reactor to tuberculin was vaccinated with B.C.G. The vaccinating officer did not note the names and ages of persons vaccinated. On being insisted to do this, his reply was that he had no instruction to keep any record in case of mass vaccination.

There is no doubt that W.H.O., which is not manned by "frivolous men", has sponsored the B.C.G. programme in various countries, as stated in your edi-

torial. But does it endorse not to keep records of persons vaccinated with living strains of tuberculosis bacilli? Another question naturally crops in the mind of right thinking man is that how did our popular Union Health Minister, arrive at the figure of B.C.G. vaccinated persons, in the absence of records? I am etc.

Suri, Birbhum. (Drs.) Kaligati Banerjee and Guruprasanna Mukherjee.

Cardiolipin in the Serology of Syphilis

Sir,—A recent communication runs: 'Cardiolipin Production: The plant at Calcutta is now in production and it is hoped that the entire requirements of VDRL antigen for the S.-E. Asia region will come from this plant' (*Daily Press*, 1955).

Ran a communication, late last year, from an international Expert Committee on Venereal Infection: '(3) The Expert Committee on Biological Standardization was not able to establish International Standards for cardiolipin and for the lecithin in the manner applied to Biological substances, as the former may have a shorter lifetime (chemical or serological) than the latter' (W.H.O., 1954).

On lecithin runs an observation: 'Further work is indicated to prepare a single lecithin . . . . the criteria for the standard calls for an iodine number of 75 to 85' (Sherwood, 1951). Obviously lecithin which is used with cardiolipin in the preparation of the antigen is not a constant chemical.

Your correspondent wrote: 'Further, limitations of the older antigens are known while those of the cardiolipin antigen must await investigations. A serologist using the latter alone incurs a serious risk and legal liability' (Greval, 1953). The morbid states responsible for the limitations are many in the tropics.

Have the workers in Calcutta considered the adverse opinions on the antigen they are preparing for S.-E. Asia? I am etc.

166, Lower Circular Road,
Calcutta - 14. S. D. S. Greval.

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REVIEWS

Erythromycin—Antibiotics Monographs, No. 1.—By Wallace E. Herrell, M.D., M.S., F.A.C.P. Pp. viii+56. Published by Medical Encyclopedia Inc., New York.

This is the first of a series of monographs that will be published on the subject of antibiotics. Erythromycin, although not as much known in this country as penicillin, streptomycin, chloramphenicol, oxytetracycline or chlortetracycline, is one that has been recently introduced. Dr. Wallace E. Herrell is not only a pioneer in the antibiotic field, he is also one of the outstanding authors in the subject of chemotherapy, as such one would find in this book everything one would like to know about erythromycin. There are nine chapters, four devoted on the subjects of history, antimicrobial activity, pharmacology, methods of assay, and methods of administration and dosage, the rest deal with the clinical or therapeutic aspects. The monograph deals with the use of erythromycin when some of the well-known antibiotics fail to control infection and the several dosage forms in which it is combined with other antibiotics. This monograph therefore will be welcome by all progressive clinicians.

Principles and Practise of Antibiotic Therapy.—By Henry Welch, Ph.D., in collaboration with others. Pp. xix+609. Published by Medical Encyclopedia Inc., New York.

In the modern age of antibiotics, any book exclusively dealing with the subject in all its aspects will be a welcome addition to the list of those already published. Welch's book is unique in the sense that it is the latest one, and deals exhaustively with all the antibiotics that are not only being demanded for therapy but even those that have possibilities for the future. The chemical, pharmacological and the clinical aspects have been elaborately dealt with. Of special value is the information given about absorption, excretion, toxicity, doses and dosage forms, which in our opinion will answer many of the queries which confront the clinicians in routine practice. Ten chapters are devoted to the individual antibiotics in the order of their discovery, in addition to the one on the isolation and development of antibiotics. A chapter by Hobby on synergism, antagonism and hormesis is as illuminating as it is instructive. The problem of development of drug resistance has led to many speculations about the necessity of combined therapy. Hobby has dealt in a masterly way a subject which has far reaching significance for future therapy. The growth stimulating property of antibiotics or hormesis is a new augury in the field. Plants and poultry seem to respond to infinitesimal amounts of antibiotics in the matter of growth, this effect on human nutrition has yet to be seen.

Eleven chapters giving details of clinical uses of the antibiotics in different types of human infections have been individually written by eminent authorities on the subjects. They have been very well classified and arranged and cover almost all the important human infections caused by bacteria. In addition special chapters are devoted to subjects like antibiotic therapy in surgical infections, ophthalmology, pediatrics, oral surgery and dentistry. We only wish there had been a chapter on assay methods, valuable as it would have been to those working in laboratories or interested in researches on problems connected with antibiotic therapy.

The book, we believe will serve as a book of reference for the practitioners.

SUPPLEMENT
Journal of the Indian Medical Association

Vol. 25, No. 12 CALCUTTA November 16, 1955

VIII ORISSA STATE MEDICAL CONFERENCE, CUTTACK

The 8th Orissa State Medical Annual Conference was held on the 10th and 11th September 1955. Delegates and members of the profession attended the Conference in large numbers from different parts of the State. Dr. J. Dei, was the Chairman of the Reception Committee and Dr. S. C. Das presided. Hon'ble Chief Justice L. Panigrahi was the Chief Guest, Sri Satyapriya Mohanty, Minister, Health, Law and Home inaugurated the Conference.

There was a Scientific Session in which Dr. B. Patnaik and Dr. S. C. Das presided. The Scientific Exhibition was opened by Dr. K. N. Misra, President, IMA, Orissa State Branch.

Dr. J. Dei, The Chairman of the Reception Committee

Dr. J. Dei accorded a hearty welcome to the delegates and guests and in her address described in short the importance of Cuttack, with its achievements past and present. Enunciating the functions of the medical profession she said, "Being the custodians of a better standard of health for our people, the basic essential for any association like ours is to see that we resolve to put forth what is best in us with regard to our services rendered to all humanity while serving our people in this State."

Dr. K. N. Misra, the President of the Conference in course of his address said,

"Health problem of our country is varied as its climate in the different parts. Each State in our country is the master of its own health plan. The Central Authority has only the advisory capacity. This system of health administration has its advantages and disadvantages also. Our inter-state relation in health matters is meagre and proper co-ordination amongst states becomes difficult. Resources of different states are also different. Hence an undeveloped state hesitates to put an ambitious health plan into effect until financial help from the centre is assured. To make India one unit, it is the first duty of the Centre to develop these undeveloped and partially developed states to the level of the developed states so that each state would make the same progress and should not feel left out.

Dr. K. N. Misra, The President of the Conference

Most of the inter-state jealousy will disappear as soon as this policy is strictly followed at the centre. The second five year plan is in the making and I hope that this point will receive due consideration during distribution of the central resources for the states for future development.

Rural Health

India is a land of villages and until we have guaranteed medical aid, preventive, curative and environmental

to these villages, our health planning is defective and inadequate. We all know how small is our progress in this direction in the first five year plan, which is nearing completion. It has failed to fulfil its obligations not so much due to lack of funds but due to inefficient administration under the plea of shortage of trained personnel.

Type of Medical Aid.

There are so many systems of medicine practised in this country, viz. Allopathy, Homoeopathy, Kaviraji, Unani, Nature cure, Divine Cure, Bio-Chemic and so on. In addition to these, quackery runs rampant in rural area due to lack of proper medical aid and ignorance of the people. A sickman in the village feels helpless and accepts the advice of any man who offers him some hope of recovery, may he be a quack, a diviner or a nature curewallah. We have no statistics to show how many men die due to such treatment in comparison with those who die for want of treatment. Curiously enough, our medical statistician is still the village choukidar. Human life is not so cheaply valued elsewhere.

Allopathy, though most scientific is rather expensive in the eyes of the villager. But cure of the disease is only a small part of our health-plan. We have to lay stress in the prevention of the disease and in the creation of healthy environment so that each individual enjoys positive health and physical fitness. We have no difference of opinion to follow the Allopathic system in the prevention of the disease and in creating healthy environment by Allopathic Drugs and Chemicals. We are committed in our health-plan that preventive and curative sides of medical aid should both be amalgamated so that a doctor can look after both sides at the same time and develop an outlook more to the need of the community than to the individual. Then will it not be very economical if we follow the same system of medicine in both curative and preventive sides? Again in the curative side, Surgery of the Allopathic system stands no competition. It is only the use of drugs in diseases that is under controversy. Why not make a thorough analysis of the drugs and the methods of their use in the different systems of medicine by the assistance of a conjoined board of doctors of each system to find out the truth in their efficacy? Allopathic drugs are however getting cheaper day by day and will become still cheaper when the pharmaceutical industry grows more in our country.

Medical Education.

A Medical Practitioner now-a-days is not only called upon to cure a sickman but also to prevent sickness in the family and in the community. He has to study also the local condition and give necessary advice for health, social, physical and mental. Hence the training of the doctor must change according to the present conception of medical aid. The Indian Medical Council as well as the Government of India are planning to call a conference of eminent teachers of medicine to draw up a suitable curriculum of study in the Medical Colleges. There has already been a world conference for the change of medical curriculum. It is not possible and not necessary to produce an internationally uniform medical curriculum, provided certain fundamentals are always observed, the practice of medicine must be as variegated in its trimmings as the flags of the nations of the world. In the training of the basic doctor, the scientific, pre-medical and preclinical courses should be more applied on clinical standpoint and details should be left out for special study. The clinical course should be more integrated than at present.

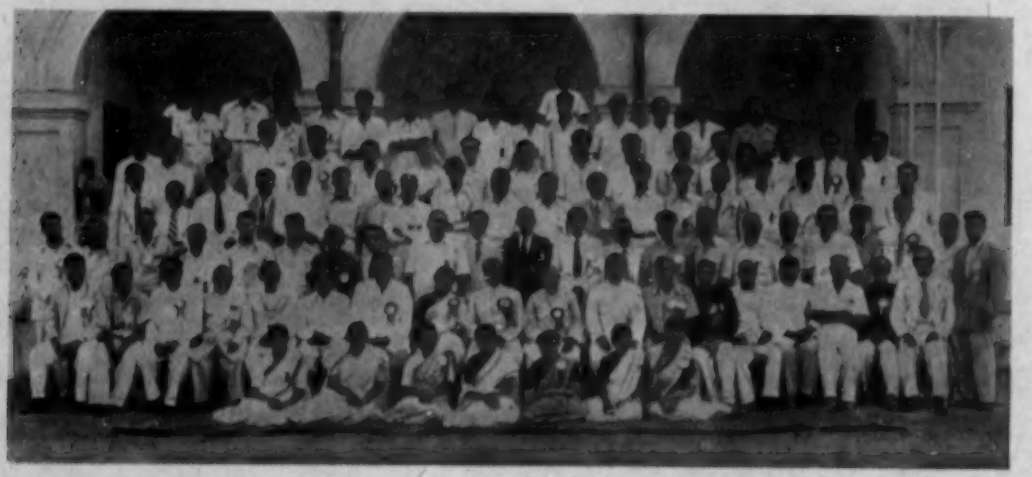
Medical Research.

The Allopathic system of medicine is regarded in this country as foreign only because most of the researches in medical science and inventions of new medicines are being done elsewhere. We practise this system of medicine with the knowledge mostly derived from other countries without contributing our own quota to the progress of the science. This state of things cannot go on for ever.

It is now time that groups of our young doctors with aptitude for research, should be trained up to carry on systematic research in different branches as wholetime workers. Adequate funds should be allotted for this purpose even though the outturn of the benefit may not be apparent in the beginning. Nature has provided this country with innumerable medicinal herbs and chemicals and it is upto our research-workers to unfold the unknown treasure for the benefit of the humanity.

Care of the old

Care of the old is a part of the social medicine in addition to woman and child welfare and care of the deaf, dumb and the mute and of the insane. No active



THE PRESIDENT AND DELEGATES OF THE 8TH ORISSA STATE MEDICAL CONFERENCE

steps have been taken in this matter in the first five year plan. With the rise of average expectation of life on better health plan, there would be many old people in the country who would require special care and rehabilitation. The best yard-stick to measure the stability of an order in society in relation to the times and conditions in which it finds itself, is the happiness of its old people. They deserve an honoured place in society they have helped to build and they are entitled to the respect and help of the generation they have bred and trained.

Medical Practice

Medical practice in this country is mostly limited to the urban area as a doctor can hardly make a satisfactory living in the villages, which are mostly inhabited by the poor agriculturists. We often tell them to be rural-minded but advice has a value only when it is practical. A doctor cannot settle himself in a village unless a certain amount of income is assured either from State or private sources. Most of our rural dis-

DR. GEORGE PATNAIK, THE ORGANIZING GENERAL SECRETARY OF THE CONFERENCE

pensaries are very inadequately provided with medicines and instruments with the result that a doctor there cannot show more efficiency than a village-quack. The latter has no medical ethics to follow and no restriction for professional advertisement and so he scores amongst the illiterate mass. Although we have very inadequate number of doctors in comparison with those in other countries on population basis, we have no satisfactory plan to mobilise them to the best advantage of the country. There are many doctors who are practically unemployed and cannot earn a satisfactory living, not because they are idle and foolish, but because they have no means to settle in a locality without financial aid from Government or private sources. It will be useful if statistics of such unemployed doctors are taken and they are rehabilitated soon.

There is a tendency now-a-days amongst our colleagues to be too much fussy in medical practice. We should fit in to the poor economic condition of our people and devise simple methods of treatment without loss of efficiency. You feel uncomfortable sometimes to work in a dispensary or clinic without the number of instruments and appliances you have seen in your teaching institutions. But you cannot carry the equipments of a medical college into a rural dispensary. We should, therefore, learn how to work with the practicable minimum. Doctors who are practising in rural areas, should see the working of Missionary Hospitals situated generally in the interior of the country and see how they perform 'high class' surgery with a few instruments and appliances, occasionally obtaining some special instrument on loan from the nearest big hospital. Much of our unpopularity in the rural area is due to the fact that we become too fussy and too expensive, beyond the means of a poor villager. So he runs to a quack. A case of pyrexia or a case of cough or chest pain or even a case of fracture should be diagnosed by you by your clinical sense. If you advise him to get the blood or sputum examined or to run to a big hospital for X-ray examination before you can diagnose his trouble, he cannot afford to get these done and will certainly run out of you. I do not mean by this statemnt that these examinations are not necssary and should not be done where facilities are available and where the patient can afford for them but I do not like unusual emphasis in those supplementary examinations over the clinical one, which only gives the real clue to correct diagnosis.

DR. G. B. MOHANTY, JOINT SECRETARY AND TREASURER OF THE CONFERENCE

The wide understanding and sympathy which are associated with good medical practice will be re-inforced if we see life as a whole and see it clearly through an upholding faith. The basis of this faith is entirely for individual decision. In these troublesome times, when mankind is finding itself implicated in so many problems of life, it is natural that the individual citizen should be able to turn to his doctor for advice on the problems of his conduct, the solution of which would mean so much to his healthy living. It is only the doctor with a philosophical trend of mind and a culture of deep sympathy for humanity, who can give that help that his patient needs.

The art of healing is not confined now to bring about only the physical fitness of the patient but also the cheerfulness and happiness of his mind. A medical practitioner has to become both a scientist and a philosopher.

Our Own State Affairs.

A medical observer who once toured the different parts of Orissa casually remarked that Orissa is the museum of Pathology. In my opinion, he made a really

true statement. But who made this and how? How could the land which possessed so many healthy men to fight out their independence up to the middle of the reign of Akbar, be so easily converted as specimens of the museum of Pathology? I am not a student of history but what little I know of, I consider the downfall of the health of our people happened due to alien rule and to dismemberment of the state by partial conquests causing fall of the economic condition. Whatever it was, it became still worse in 1866 when people died in millions like cats and dogs. Many people ran to shelter homes for food and care. We lost our national culture due to poverty and became a backward people in comparison with those of other parts of India. Our real administrators then remained in distant places, portions of our state being tagged to other states in the neighbourhood. We were actually governed by our own brothers, who did not consider this part of India as their own home and exploited us. We lost our wealth, health, courage and even our culture. We had to run to distant parts of India mostly as labourers to earn our livelihood. The state remained dismembered and mother 'Utkal' became 'Chhinnakaya'. The Oriya who are found in Burma, Assam Tea Gardens and Calcutta industrial area

DR. M. C. MISRA, SECRETARY,
ORISSA STATE BRANCH, I.M.A.

have not gone there on pleasure-trips but in utter necessity to find out a living, as their own state is unable to provide them with employment to improve their economic standard. How can one find positive health in a people with such social and economic back-ground? If this part of India has to progress equally as any other part, the low level in the economic condition, education and health should first be brought up to the level of other states by extra grants and subsidies. If national health service and free medical aid are ever planned for India, it is this part which deserves the first attention. The people here are displaced persons in their own houses and require rehabilitation. Here we are poor not by our own choice but by historical circumstances already mentioned. The museum of Pathology has to be changed to a museum of Physiology and it is a central responsibility. Ours being an ill developed state, we naturally expected a lot of improvements in the first five year plan. We know now how little progress we have made. With a population of 1,46,45,946 in 1951 and probably a little more by now, we have opened or are going to open, as far as my information goes, only 26 additional dispensaries, 10 medical-aid centres, 12 Ayurvedic dispensaries and 12 Health centres in 'Community Project' areas. We have started six anti-malarial centres to serve a population of 60 lacs, an anti-filarial unit to serve about 30 lacs of people. We have made some progress in the B. C. G. campaign against tuberculosis, the utility of which is still questioned in some quarters. Is this list of health activity sufficient for a period of five years? I am afraid if we continue to make progress at the same rate in the next five years in our health plan, half of our population will vanish by the time the third five year plan begins.

Orissa is a rich soil for hook-worms and probably half of our population in the villages suffer from the infection. Lethargy is one of the symptoms of the hook-worm disease and it is no wonder that our people are proverbially lethargic because of this infection. What are we doing for this? Leprosy has a high incidence in our State and for this, Government of India wanted to establish a research centre here for the particular disease. But it has now passed on to another state on account of slackness of our administrators. Leprosy drugs are cheap now. Should we not arrange compulsory treatment of all lepers in our state, to eradicate the disease completely? Our only medical college at Cuttack is still unequipped. It has no up-to-date lecture theatre or demonstration rooms. Residential quarters are so insufficient that even the staff doing emergencies come from outside. The teaching staff are called outside urgently to attend on members and officers of Government even during their hospital rounds or class-hours, and I understand that they sometimes are asked to explain if they are late by a few minutes. How long would this state of affairs continue in our only institution and what lessons would our would-be doctors carry for their noble service?

We have accepted the view that the medical service and the health service should be integrated into one on the recommendation of the Bhore Committee. What have we done for this? The two services are as separate as before, in tight compartments and sub-compartments. It was accepted by Government that all provincial services should have the same scale of pay. A medical officer was not granted that privilege on the plea of income from private practice. Although the service itself wanted to work without private practice, the request was not heeded. Even the health officers who have no private practice, are not allowed the usual scale of the provincial service. Why make a principle and then break it?

Towards the end of the first five year plan, with a population over 1,46,45,946, we have only 1222 registered doctors, i.e. one doctor for a population of 11,985. It will be interesting to know that even Assam with so much flood and earth-quake has now 3,000 registered doctors for a population of about 92 lacs, i.e., one doctor for 3,000 population. The figures for nurses and midwives are still worse. We have only 228 registered nurses, i.e., one nurse for a population of 64,237, and 291 registered midwives, i.e., one midwife for 50,330 people. What are we doing to train up more health personnel?

I am not aware of the details of the second five year plan for health but I am afraid, as I learn from papers, it is also not bold and comprehensive. We have urgent need of post-graduate studies in our only medical college for future making of teachers and for research. We have urgent need of a big hospital, say with at least 750 beds in the industrial area near about Sambalpur so that another medical college can gradually grow there. We have urgent need of a hospital of Infectious Diseases at Cuttack, for proper segregation of cases from the town and for proper teaching of students. We have also great need of a Mental Hospital in these days of stress and strain. We have great need of health centres in the rural area, say, at least 20 in each district. We

have urgent need of control of enteric fevers and cholera by compulsory vaccination as for small-pox, in addition to control of malaria, tuberculosis, hookworm disease, leprosy and filaria. We must move fast, plan fast and work it out fast.

Our Association.

This Association has to take greater part in the health programme of the country. Unfortunately Government do not show the respect and sympathy it deserves. It is always ready to co-operate in every field of activity of the health programme of the State and give useful advice. In the national up-building of the country, we have a major part to play in social welfare and health development.

Your Association is what you have made it. No work can be pursued unless there are people to do it with pleasure and sincerity. Unfortunately, many of us want the association to pay dividends only and leave aside the value of social contact and comradeship in profession. So we are unable to work in a systematic plan.

Membership of our Association is far from satisfactory. There are only 172 members on the roll. Some districts have not even been aware of the activities of the Association. Such apathy and such outlook only lower you in the eyes of your colleagues and make you barren of social interest and scientific knowledge. Social contact is a vital force which enriches your knowledge and outlook day by day and once you lose it, you lose interest in your life. I request you all who are not yet members of the Indian Medical Association to join it soon and make it strong and fully representative.

We are now in the midst of a terrible catastrophhe. Nature has been very unkind to us this year. Thousands of villages in our State are submerged in flood and we have no idea of the extent of the damage and loss of life. Thousands of our people are without food and shelter. Several cases of snake bites are already reported. Soon after the flood, severe epidemics of Cholera, Dysentery and enteric fever will break out, which are now-a-days preventable provided timely action is taken. In this calamity, Orissa expects every one of us to do his duty. The doctors and medical students have an important role to play in the prevention of these epidemics. The State Branch of the Indian Medical Association should organise a volunteer medical corps and should co-operate with Government in looking after the health of these unfortunate people without delay. Here is an opportunity to save and to show the worth of our Association. I hope, my appeal will have sufficient response.

The following resolutions were passed at the Conference :

1. Resolved that this Conference views with great concern the devastations caused by the widespread flood in the coastal areas of this State and forms a Volunteer Medical Corps for giving Medical Relief to the flood affected victims. The money saved from Conference Dinner would form the nucleus of the fund for the organisation.

2. Resolved that the Government be requested to follow the example of West Bengal in giving special allowance to the medical employees in rural areas as follows—for Graduates, Rs. 200/- p.m., for Licentiates, Rs. 100/-p.m.

3. Resolved that the Government be requested to form an Orissa Medical Code as soon as possible.

4. Resolved that the Government be requested to take immediate steps for grant of concessions promised to the ex-service personnel of the Army Medical Corps.

5. Resolved that to safe-guard the interest of the medical profession in case of professional mishaps not due to the negligence of the doctor, the doctor should not be hauled up by the Police without approval of competent medical authority. (Standing Medical Tribunal consisting of three members).

6. Resolved that this Association move the Government to take adequate steps to stop quackery in the Indian Union.

7. Resolved that National Health Service being the final aim, the Government may be approached to abolish the saletax on the sale of medicines and medical accessaries as immediate step to ease the availability of the medicines to the poor.

8. Resolved that this Conference recommends to the State and Union Governments to take earliest possible measures in instituting an All India Cadre for teachers of the Medical Colleges in a broader interest of raising the standard of medical relief equally all over the country. This will also enable the medical teachers to encompass a progressive outlook by working in contemporary Teaching and Research Institutions existing in the country.

9. Resolved that in the interests of National Health, this Conference urges upon the State Governments, University and Public Bodies to take measures as a priority programme to provide suitable open spaces, play-grounds, gymnasia, etc. all throughout the State of Orissa for uplifting the standard of physical health of our people.

10. Resolved that this Conference strongly urges on Government to take immediate steps for changing the rules and regulations of the Orissa Council of Medical Registration so as to make it fully representative by replacing the nominated members by members duly elected from amongst the registered medical practitioners of Orissa.

11. Resolved that this Conference is of opinion that the Ayurvedic School at Puri be converted into an Ayurvedic Research Institute.

12. Resolved that this Conference requests the Government to seek co-operation of the Medical profession by inclusion of representatives of the Indian Medical Association to the Development Committee.

13. Resolved that the State Government be requested to arrange periodical refresher courses at the S.C.B. Medical College, Cuttack for the general medical practitioners at the State expense.

I.M.A. QUESTIONNAIRE ON MEDICAL EDUCATION

The Indian Medical Association Medical Education Sub-Committee has issued a Circular dated 27-9-55 to Hony. Secretaries of all State/Territorial Branches of the I.M.A. regarding I.M.A. memorandum on Medical Education in connection with the First World Medical Conference on Medical Education and the Second on it to be held in 1959 in U.S.A.

The Circular reads :

The Indian Medical Association has been requested by the World Medical Association to present its considered views on the subject of Medical Education by March, 1956.

The following proforma has been drawn up by the IMA Medical Education Sub-Committee to collect the opinion of the different State and Territorial branches on this subject.

The subject has been divided into 4 sections.

Section I : Requirements for entry into Medical College.

Section II : Aims and content of the Medical Curriculum.

„ III : Techniques and methods of Medical Education.

„ IV : Social and Preventive Medicine.

You are therefore requested to send the opinion of your branch in the way suggested by the 30th November, 1955 at the latest to the Convener of the Sub-Committee C/o. The Bengal Provincial Branch of I.M.A. 67 Dharamtola Street, Calcutta 13.

Apart from the items mentioned above any other relevant information or opinion if considered necessary may also be kindly included in your report.

While replying to the questions, you are particularly requested to number the replies in the order the questions appear in the proforma.

SECTION I

(Requirements for Entry)

1. (a) What should be the correct level of general education for admission into Medical College?
   (b) In view of the proposed abolition of the I.Sc. and the change over to the three year degree course what are your views on :—
   Accepting the pre-university course as satisfactory for admission into the Medical College. Introduction of a year of pre-medical study after the pre-university course,—in a Medical College or in a college of Science.
   Prescribing a pass in the 3 year B.Sc. course (with physics, chemistry, biology, statistics and psychology) as minimum qualification for admission to the Medical College.
   (c) Should a minimum age be prescribed for admission to Medical College? If so what should be the minimum?
   (d) Do you consider a liberal and general education, which would include such subjects as History of Medicine, broad education in the humanities (literature, history, politics, sociology) social relations of science etc. is necessary to make the student more able to grasp the importance and implications of the medical and allied subjects he is required to learn later? If so at what stage would you think these subjects should be introduced?
2. What are your views on the value of the following in the selection of a medical student.
   (a) Competitive examination and interview for entry,
   (b) Personality, intelligence and aptitude tests
   (c) A Specific report from his teachers in the general educational institutions where he had studied.
3. How much Physics should he know? Since application of physics is necessary for diagnosis, therapy and the study of fundamental medical problems, do you think it should also be taught during the clinical period in collaboration with a teacher of internal medicine?
4. How much Chemistry should he know? Should the teaching concentrate on the understanding of the structure and properties of these substances which occur in the body and take part in metabolic processes and also the structure of drugs and substances used in therapeutic agents?
5. How much Biology including evolution and genetics, should he know?
6. What is your opinion on the question of encouraging entrants from lower economic groups or any social groups in larger numbers by State subsidy?
7. Do you think more merit stipends should be instituted?
8. What is your opinion about reservation of seats for women?

Kindly indicate the existing requirements for entry into Medical College in your area.

SECTION II

(Aims and Content of the Curriculum)

1. What in your opinion should be the aims and content of the medical curriculum. Will the sole object be the production of a general practitioner or laying the foundations of a medical carrier, whatever branch of medicine may later be followed?
2. How much of Anatomy should the doctor know, and at what stages? How much detailed knowledge of the human body is necessary for the practising doctor and that for developing into a Surgeon?
3. How much applied Anatomy should the doctor know and at what stages?
4. How much Physiology should the doctor know and at what stages? Must teaching stress on principles rather than on details? Are the students being overloaded with facts? How much applied Physiology should the doctor know and at what stages?
5. (a) Do you think integration of Physiology and Anatomy as a single subject or correlation between the two in the curriculum will be more advantages to the students? If so how could this be introduced in the joint course of lectures?
   (b) If integration of anatomy and physiology is not favoured what do you think should be the position of histology with anatomy or with physiology?
   (c) What should be the position of biochemistry—as an independent department or integrated with physiology?
6. (a) How much Pathology should the doctor know and at what stages? Do you think it is being finished too early and is being pushed back upon the museum and the microscope and pathology is being confused with morbid anatomy by students as well as by teacher?
   (b) Is a reduction in the course of pathology from 2 years to 15 months desirable? If so what arrangements should be made to teach special pathology?
   (c) How much bacteriology should be taught? Should bacteriology be a separate department or integrated with pathology.
7. How much pharmacology should the doctor know and at what stages should this be taught entirely, chiefly or only partly in its applied aspect?
8. What in your opinion should be the place and scope of the following subjects in the medical curriculum?
   (i) Pediatrics, (ii) Psychiatry, (iii) Tropical medicine, (iv) Industrial Medicine, (v) Social and Preventive Medicine, (vi) Experimental Medicine, (vii) Minor Surgery, (viii) Elementary Medical Statistics and design of experiments.
9. Is an interim year, year of practical training necessary before registration?

Kindly indicate the curriculum in your area.

SECTION III

(Technique and Methods of Medical Education)

1. How would you like the following constituents of medical teaching to be conducted?
   (a) Hospital bedside teaching in medicine,
   (b) Hospital bedside teaching in surgery and in the theatre,
   (c) Hospital teaching in the out patients.
   In considering the above three it would be useful to indicate the value of didactic and demonstration procedure and their relative proportions. Since practice conditions are more closely simulated in the out patient department, what definite scope should be provided in this respect to the students?
   (d) Home teaching :—Do you think that it will be useful for the senior student to utilise sometime in visiting patients in their homes as a part of a "Home Care" medical team consisting of residents, social workers etc.?
2. What are your views about the usefulness and the mode of organisation of—
   (a) Clinico-pathological Conferences,
   (b) Clinico-Sociological Conferences,
   (c) Theoretical Lectures,
   (d) Demonstrations,
   (e) Group discussions.

3. Is it necessary to teach the teacher to teach? If so, how?
4. What are your views on the formation and usefulness of—
   (a) theoretical papers, (b) practical and oral tests?
   Are both necessary, if so in what proportions? Is it possible to dispense with any of these?
5. Why students fail?
   Considering the fact that mostly the best students are admitted in Medical College do you not think there should be very few failures to qualify in the first chance? If the answer is Yes, what is the remedy?
6. What are your views on the usefulness and feasibility of the following as methods of teaching—
   (a) Motion pictures,
   (b) Visual aids,
   (c) The still picture,
   (d) One way observation screen,
   (e) Laboratory training in the teaching of Experimental Medicine.
7. What is your view on the existing methods of Examination? Would you like them to be modified? If so in what way? What is your idea of introducing seminar system of examination—its importance vis-a-vis the University Examinations?
8. What are your views on the mode of organisation of the library and reference services?
9. What is your opinion on the employment of the full-time system in Medical Education?
10. What do you think should be the place of research in undergraduate institutions? Considering the fact that an understanding of the scientific method, which should be an important aim of education, is gained only after exposure to a problem, do you think, with advancing experience students should be given the opportunity of designing experiments?

Kindly indicate the existing conditions in your area.

SECTION IV

THE TEACHING OF PREVENTIVE AND SOCIAL MEDICINE

1. Since the Clinician or General Practitioner of to-day is expected to practise both preventive, cure and rehabilitation diseases and promotion of health, many of which are related to social, economic and environmental factors and since the General Practitioner forms the primary link with the patient in his home and community, it is necessary that the medical student should be taught to look at medicine as a social science and to determine the interconnections between health and disease on the one hand and biological, social and economic differentials on the other.
   If you agree with this statement, do you think the integration of teaching, with related subjects, should permeate the whole curriculum or only the clinical period or a part thereof.
   The present curriculum in the subject of preventive and social medicine in most Indian Universities arranges for only 1-2 year's training. Do you think this is adequate for the purpose? If you don't think so, would you suggest how best the curriculum can be spread through the clinical period?
2. In view of the Clinical approach to a public health subject and the need for collaboration with other departments and the community, do you think the teacher should be a Public Health Worker or a Clinician with a public health outlook and interest?
   Do you think a part-time Professor, as it prevails now in most Medical Colleges, can adequately impart training in the subject or do you prefer a whole-time Professorship for the subject?
3. Since the bulk of the students are expected to go into general practice, do you think a Health Centre with domiciliary care should be attached to a teaching hospital, in order to permit clinical clerkship in families and to impart more practical training?
   If there is no such Health Centre attached to a teaching hospital, how do you think effective teaching in the new concepts can be imparted?
4. Do you agree that when a general practitioner sends a patient to a hospital for diagnosis and treatment, that patient, on discharge, should be returned to the general practitioner with relevant notes and suggestion and that this practice will influence the social outlook of the medical student for follow up, rehabilitation and other problems?
5. Since Sociology is still a problem dealing with man as a biological creature at what stage in the student's career do you wish that some training in sociology (including anthropology, economics and ecology) should be imparted?
6. As regards the relationship of working environment to disease, how much of industrial hygiene, the modification of industrial stress and the actual work of industrial medical practice should be taught to the undergraduate student?
7. Do you think the student health service of the Medical College and the University should form a part of a department of Preventive or Social Medicine?

BRANCH NOTES

AHMEDABAD BRANCH—The annual general meeting of the branch was held on 24-9-55. Office bearers for 1955-56 were elected with Dr. R. A. Desai as president and Dr. A. P. Shukla as hony. general secretary. Dr. R. K. Nanavaty and Dr. B. M. Joshi were elected jt. secretaries.

AHMEDABAD MEDICAL SOCIETY—The annual report of the society for the year 1954-55 shows that 32 new members were enlisted during the year, total membership coming up to 336 including 2 life members. The following meetings were held :—Clinical meetings—26, Ordinary meetings—3, Special meetings—4, Extraordinary meeting—1 and Managing Committee meetings—16. Regular scientific meetings and discussions were held. The subcommittees met regularly and discussed matters referred to them. The 6th All India Industrial Medical Conference was held in this city in February 1955, the members of the society taking active part in it. The financial position during the year appears to be satisfactory.

ANANTAPUR BRANCH—The annual meeting of the branch was held on 24-9-55. Office bearers for 1955-56 was elected with Dr. G. Thimmareddi as president and Dr. V. K. Rajaram Rao as secretary. Dr. P. T. Sreenivasan was elected jt. secretary. Dr. D. R. Manjappa of Minto Ophthalmic Hospital, Bangalore read a paper on "Use and Abuse of Antibiotics in diseases of the eyes". The annual report of the branch for 1954-55 shows slight change in membership. Eight meetings were held during the year. The financial position was satisfactory.

ARARIA BRANCH—The annual general meeting of the branch was held on 24-9-55. Dr. B. Taheed presided. The annual report for 1954-55 and the audited accounts were adopted. Office bearers for 1955-56 were elected with Dr. N. K. Mehra as president and Dr. L. M. Kurdoo as hony. secretary and Dr. Md. Siddique as jt. secretary.

BHIR BRANCH—A meeting was held on 19-9-55. Dr. Gulam Nabi was elected president for 1955-56 and Dr. H. R. Kandlikar, hony. secretary. Eleven members were present. Dr. Gulam Nabi spoke on cases of Intestinal Obstruction, Volvulus and Ticck Typhus. Dr. Sham Rao demonstrated a case of Lymphatic Cyst in front of neck. Dr. S. N. Deshpande read a paper on Vitamin B_{12}, its pharmacology and therapeutics.

BIHAR-SHARIF BRANCH—At the annual meeting of the branch held on 14-9-55, Dr. R. L. P. Varma was

elected president and Dr. M. M. Haque, the secretary of the branch for the year 1955-56.

BIKANER MEDICAL ASSOCIATION—The annual report for 1954-55 shows, that 15 new members were enlisted during the year. Thirteen meetings were held and besides official business, medical and surgical subjects were discussed and a few cases of clinical interest were demonstrated. During an epidemic of Diphtheria, the association held a symposium on Diphtheria and its prevention. A few scientific films also were shown.

COCHIN BRANCH—On 16-10-54, an ordinary meeting was held. Demonstration of Clinical cases by Capt. M. A. Urumese of General Hospital, Ernakulum was held.

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On 22-10-54, a special meeting was held. Demonstrations of two talkie films on Aureomycin and Polvite were held.

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On 27-11-54, an ordinary meeting was held. (1) Address by S. B. Rao on "The Birth of a new drug" and (2) address by Sri V. R. Sastry on "The Problem of our fallen Sisters" were given.

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On 21-12-54, an ordinary meeting was held addressed by Dr. C. O. Karunakaran, President, Travancore Cochin Medical State Association on "Employees State Insurance".

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On 12-1-55, an ordinary meeting was held in which Prof. R. Rothlin, Director, Pharmacological section of M/s. Sandoz Ltd., spoke on "Modern Conception of the Autonomic Nervous System and its clinical importance."

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On 29-1-55, a special lunch meeting was held to give a most befitting welcome to Dr. S. C. Sen, President, Indian Medical Association.

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On 12-2-55, an ordinary meeting was held in which Dr. George Coelho, Hony. Pediatrician, Byramjee Jeejeebhoy Hospital for Children, Bombay spoke on "Convulsions in children".

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On 10-3-55, an ordinary meeting was held when demonstration of clinical cases by Dr. P. K. Raghava Warrier, was held.

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On 12-4-55, in an ordinary meeting of the branch a demonstration of clinical cases was given by Dr. P. K. Raghava Warrier.

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On 9-5-55, an ordinary meeting was held which was addressed by Dr. R. G. Ginde, Hony. Surgeon, King Edward V Memorial Hospital, Parel, Bombay and Beach Candy Hospital and Nursing Home, Bombay on 'Brain Tumour'.

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On 10-6-55, an ordinary meeting was held in which a demonstration of clinical cases by Captain M. A. Urumese and Dr. N. Vasudevan was given.

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On 19-7-55, an ordinary meeting was held in which abstracts from leading medical Journals of India and abroad and demonstration of clinical cases were read.

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On 16-8-55, an ordinary meeting was held in which 2 articles of Drs. J. C. Patel and R. G. Ginde were read and clinical cases were demonstrated.

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On 7-9-55, a special meeting was held to give a most befitting welcome to our professional brethren from various provinces of India undergoing a special course at the newly started Filaria Control Centre, Ernakulam. Dr. A. K. Krishnaswamy, Asst. Director spoke on 'Filariasis, Past, Present and Future' and Dr. N. G. S. Raghavan on 'Filariasis'.

• • • • •

On 19-9-55, an annual meeting was held in which Office bearers for 1955-56 were elected with Dr. S. S. Rao as president and Dr. B. D. Mallaya as secretary, Drs. A. P. Sebastian and P. V. George were elected joint secretaries.

VI ALL HYDERABAD MEDICAL CONFERENCE

(Under the auspices of the Indian Medical Association)

The 6th All Hyderabad Medical Conference is taking place in Gulbarga on the 19th and 20th Nov., 1955.

This conference will be a focal point for Physicians, Surgeons, Medical Officers, Health Administrators from all parts of this State. The reception committee has decided to bring out a Souvenir (Size $9'' \times 5''$) and arrange a Scientific Exhibition and a District Exhibition as well.

The Scientific Exhibition will last for 2 days simultaneously with the District Exhibition. Stalls will be arranged just adjacent to the Pandal in the heart of the town. The size of the stall will be $12' \times 10'$ and the rates will be charged at I.G. Rs. 50/- per stall till the session lasts.

XX U.P. STATE MEDICAL CONFERENCE, BAREILLY.

The 20th U. P. State Medical Conference will be held at Bareilly on the 27th and 28th November, 1955. Members of the profession are requested to write papers and read them in the Scientific Section of the Conference. Further information may be had from the Organising Secretary of the Conference, Dr. G. S. Dhillon, Pul Qazi, Bara Bazar, Bareilly.

X MADRAS STATE MEDICAL CONFERENCE, SALEM.

The 10th Madras State Medical Conference will be held at Salem on 3-4 December, 1955. The delegates' and visitors' fee has been fixed at Rs. 15/-. The Executive Committee of the Reception Committee has been formed. Further information may be had from the Organising Secretary, 10th Madras State Medical Conference, Salem.

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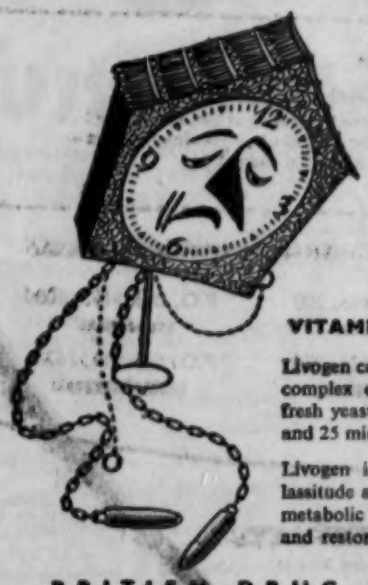

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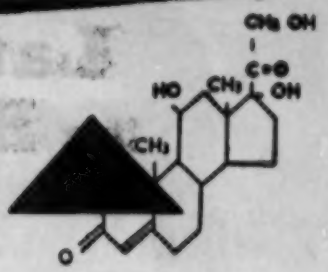

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Regd. No. C 1890 November 16, 1955

APPEAL

For The

"BUILDING FUND"

Of the Central Office of the Indian Medical Association at New Delhi

Attempts have been made for years past to secure a suitable plot of land in New Delhi for the construction of a building of the Central Office of the Indian Medical Association, and appeals were made in 1951 and subsequently to all the members of the Association to contribute generously to this Fund.

I am happy to announce that the Government of India has finally decided to allot us a plot of land measuring 0.5 acre at a concession price of Rs. 18,000/-/- plus 5 per cent ground rent. The building will have to be completed within a period of two years and will have to conform in size and pattern to neighbouring buildings.

You will recall that the Working Committee and the Central Council decided in 1950 that every member of the Association should pay a minimum donation of Rs. 50/- in one or more instalments; those who are better off are expected to pay much more.

It is proposed to have, apart from rooms necessary for the Central Office, a spacious library, one or more meeting rooms, a good sized auditorium, a decent lounge and a refectory. It is also proposed to have a few rooms for the use of members visiting Delhi for short stays either on Association work or otherwise. The total estimated expenditure will come up to Rs. 8 lacs. So far, we have received donations above Rs. 17,000/-, some members having donated Rs. 1,001/- each.

In view of the urgency of the situation, may I request every member and every Branch Secretary to kindly take active steps in the matter and send contributions either direct to the Central Office or through Branch Secretaries?

S. C. Sen

NEW DELHI,
November, 1955

President,
Indian Medical Association.